OF THEE WE SING*

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Two sentences from *The Proceedings of the Medical College of Georgia* serve as the theme of my address today:

"Those who receive the mantle of a rich and noble heritage are expected to perpetuate it."

"For those who wear such a mantle and give it no sustenance it becomes but an empty echo out of the past."

The Chattahoochee River, singing its song, flows,

> "Out of the hills of Habersham
> Down the valleys of Hall,"

By its banks lies the beautiful city of Atlanta, nestled among Georgia's red clay hills. Atlanta is a great city, a city with a heritage.

There is grandeur in the picture you see here today; great factories silhouetted against the skyline, fine buildings pushing their heads above the bustle of busy shops below and beautiful homes which dot the wooded hillsides. But it was not always thus. Eighty-five years ago there was little here of grandeur, but much of hunger, ashes and ruin. The vicissitudes of a tragic war had forced the vanquished armies of the Confederacy to retreat, while hard on their heels came the torch-bearing victorious legions of Sherman; and when Sherman left, efficient soldier that he was, he saw to it

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that everything of value to the enemy was burned to the ground. From these ashes, from a state of chaos and ruin, Atlanta, by the grace of God and American enterprise, has emerged as you see her today, resplendent in her glory, Princess among the cities of our nation.

It has been said, "A land without ruins is a land without memories; A land without memories is a land without liberty." Here in Atlanta and throughout Georgia there were ruins, and now there are memories, poignant memories of forefathers who feared the power of the Federal Government; of forefathers who fought and lost a great war on the principle of Federal versus State Sovereignty. Here, too, there is liberty and the souls of men are free, for this is America where, in the words of the great Lincoln, there exists "Government of the people, for the people, by the people."

With nostalgic reverence to a great heritage, relive with me the memories of the crucial days of 1864 and 1865. Chattanooga had fallen and Sherman, with the Armies of the Ohio, the Cumberland and the Tennessee, was pointing eastward, toward the sea. Joe Johnston, entrenched at Dalton (just 95 miles from here) was all that stood between Sherman and Atlanta. If Atlanta fell, the Confederacy was doomed, for the way to the sea would be open.

Johnston was forced to evacuate Dalton and then in rapid succession came the retreat from Resaca, the retreat from Rome, the crossing of the Oostanaula and the Etowah—then Dallas, Marietta and Kennesaw Mountain, just 18 miles away. The Chattahoochee was crossed and Johnston, in his final effort of resistance before Atlanta, entrenched his troops at Peachtree Creek. Jefferson Davis had become impatient with retreats, so, in a gesture, tragic in its hopelessness, he rushed Hood from Virginia to replace Johnston. Hood attacked, Peachtree Creek was lost, and the gates of Atlanta were open. There was great rejoicing in Washington, but in Georgia the torch and starvation did not make for joy, nor did defeat lead to happiness.

The end was in sight, but Georgia must yet be devastated. The march to the sea was accomplished with ashes and destruction in its wake. Savannah fell, then Columbia, Wilmington and Charleston. Only then came merciful surrender, Lee to Grant at Appomattox, April, 1865.

The editor of the Atlanta Constitution, Henry W. Grady, has described in immortal words the Confederate soldier as he turned his face homeward from Appomattox. "What does he find when, having followed the battle-stained cross against overwhelming

odds, dreading death not half so much as surrender, he reaches the home he left so prosperous and beautiful? He finds his home in ruins, his farm devastated, his slaves freed, his stock killed, his barns empty, his trade destroyed, his money worthless, his social system, feudal in its magnificence, swept away; his comrades slain and the burden of others heavy on his shoulders."

This was the price of war, the price of defeat—nor did the victor emerge without his dead, his heartaches and his sorrows, for in war the only thing more tragic than victory is defeat. This was the price paid by our forefathers, of the South and of the North, for having the courage and the will to fight for what each considered his rights and independence.

It took this terrible war to settle, for all time, the principle of the sovereignty of the Federal Government over the individual states. Today we all rejoice that that problem was settled and settled as it was, by the will of God. But there is another problem in relation to the Federal Government that was not settled by that war, nor has it ever been settled. It is the problem of the growing power of the Federal Government. It is the question of how large and how powerful the Federal Government may become before "Government of the people, for the people, by the people" is replaced by despotism in the form of "Government of the people, by the bureaucrats, for the bureaucrats."

You may well ask what the history of Atlanta and the problems of Government have to do with a meeting of the Southeastern Surgical Congress? I answer that the history of Atlanta, the history of the South and of the nation is our heritage, and that the problems of government must be our concern as citizens!

You and I are proud of the scientific achievements of American Medicine. You and I are proud of the American Doctor. You and I believe that medical education in the United States has no equal. But, gentlemen, do you sincerely believe the American doctor is doing his full part as a citizen? I ask: Do you believe that our medical schools are doing their full part in training students in the art of citizenship?

Have you, any of you, ever spoken to your students of responsibility to country? Have you considered the value of liberty, the danger of despotism? Have you talked of the Constitution, of Jefferson or Lincoln? We, the citizens of America, have a great heritage. God grant that we, the physicians of America, do our part in perpetuating that heritage.

That is my fervent plea today.

I beg you to remember that, in the days of the Confederation, the greatest obstacle to the union of the colonies was the fear of giving power to the Central Government. For one hundred and sixty-nine years, from Jamestown to the Declaration of Independence, the colonies had been subjected to the sovereignty of the British crown. But then came Concord, Lexington, the Declaration of Independence and the Revolutionary War. The surrender of Cornwallis was the end of British sovereignty. Where, then, did sovereignty lie? The articles of Confederation were an unsuccessful answer, for each state was to retain its "sovereignty, freedom and independence."

It did not take long for the colonists of the Confederation to see that, dangerous as it was, the Federal Government must be given more power. A uniform system of currency was a necessity. Authority to levy and collect taxes was a necessity. It must have been a hard blow to the patriots of the thirteen states to realize that their newly-won freedom must be given away, in part at least, to a Central Government. Knowing these men as we do, we could have been sure, even then, that the Constitution, which was soon to be fashioned, would protect the rights of the individual and limit the powers of government in a manner as forceful as possible for the mind of man to conjure.

On the second Monday in May, 1787, the Constitutional Convention opened in Independence Hall in Philadelphia. The Wright Brothers had not yet been to Kitty Hawk, which might explain why a quorum was not reached until eleven days later. After four months of debate, four months of acromonious debate and compromise after compromise, the Constitution of the United States was adopted.

The framers of the Constitution loved their liberty. The scars of the oppressor's heel was imprinted deeply in their souls and the surrender of Cornwallis was fresh in their memories. They had lived under oppression and tasted of liberty. They were not amateurs. They were students of Magna Carta, the First Charter of Virginia, the Petition of Right, and the Glorious Revolution in England which resulted in the first Bill of Rights. They were familiar with the works of John Locke, Baron de Montesquieu, Samuel Adams and Thomas Paine. No wonder Jefferson called them Demi-gods. Small wonder that William Gladstone said of their labors, ". . . the most wonderful work ever struck off at a given time by the brain and purpose of man."

Historians tell us that this Constitution which we so love and respect is the world's classic example of a written Constitution. Yet, Washington said of it, when speaking for ratification, "The Constitution that is submitted is not free from imperfections." But since, "A Constitutional door is opened for future amendments and alterations, I think it would be wise in the people to accept what is offered to them." The people did accept, at least after the Bill of Rights was promised, and thus began the American system of Constitutional Government.

The Constitution is not a long document. Implementation was left to the Congress. Interpretation was of necessity left to the courts. Our government has kept pace with the changing times and that, paradoxically, is evidence of the strength of the Constitution and at the same time it is evidence of the dangers that may confront us. Interpretation of the Constitution is not only a Judicial prerogative but one also of the Congress, the President, and of custom and usage. As a result, the unwritten features of our Constitution are many hundreds of times more lengthy than the Constitution itself, and many of the most conspicuous features of the American system of Government have no apparent Constitutional foundation.

The framers of the Constitution felt that they had successfully and forever limited the power and size of the Federal Government, at the same time leaving the door open, as Washington said, for future amendments and alterations, to take care of the changing thoughts in the minds of men and the growth of the country. I doubt that they, any one of them, ever envisioned in their wildest dreams the power and the size of the Federal Government as it exists today. I doubt that any one of them could have foreseen the usurpation of power by the Chief Executives of the country, and yet none other than the great Democrat, Thomas Jefferson, was the first president to set the example when he made the Louisiana Purchase without authority of the Congress, admitting at the time that he doubted his constitutional right to do so. Other presidents have gone even further in modifying and expending the Constitution—Lincoln, Cleveland, Theodore Roosevelt, Wilson, and most recently, Franklin Delano Roosevelt, carried the trend to a degree greater than any of his predecessors, despite restraint by the Supreme Court, which he attempted to ridicule by the appellation, "Nine old men."

Today we have become a Government of Bureaus. There is nothing wrong with bureaus, but, the ambitions of poor weak mortal man tends to make the bureaucrat a danger to Liberty. The rapid

growth in the number of federal employees and in the size of the annual payroll during the past twenty years demands the serious consideration of every thinking American citizen. In 1932 the total average federal employment was 621,580 and the payroll $1,059,138,000. Today the total average employment is two and a half million with an annual payroll of eight billion dollars. In other words, in twenty years federal employees have increased four hundred per cent and the federal payroll eight hundred per cent. Gentlemen, when will it end, and where will it lead? I aver that there must be a point where government, by mere force of size and power, will become despotic and freedom will be lost, and this in spite of the Declaration of Independence and the Constitution of the United States of America. Again I ask you how far the present journey can safely go?

We not only fear the present size and power of government, we fear even more the political philosophy of some of our leaders today; leaders who preach the doctrine of ever increasing size and power of the various bureaus and, mark me well, in the name of welfare and security.

We live today in the era of the Mink Coat, the Deep Freeze, Reconstruction Finance Corporation influence and Internal Revenue deflections. We do not know what the cause is of this disgraceful era in American history. Perhaps it is the mushroom-like growth of government which makes control impossible; perhaps it is a moral degeneration of the citizens of the country; perhaps it is lack of leadership; perhaps it is the normal human result of a political party too powerful and too long in control. Whatever the cause, the result is sickening.

A country is only as strong as its citizens. A country is only as moral as its citizens. Indeed a country is only as great as its citizens make it great. When the great mass of intelligent, thinking people of a nation forget their duty to their country and their government, then that country is doomed. We have today the greatest country in the world and the American Constitutional system of government has no peer. But, it is not written that this country will forever be great, nor is it certain that even our Constitutional system can forever withstand the onslaughts of a political philosophy that would make all men mere wards of the Central Government.

The physicians of America are a small but well educated, intelligent, thinking group of worthwhile citizens. To us there has been thrown a challenge and we, as a profession, have accepted the challenge. We have done well in fighting against an even larger Central Government; we have educated many to the dangers of social-

ism and despotic federal power. But we must do more. Every man of us must do his part, never forgetting that our country comes first, even before our profession. As Southerners, we might seriously weigh the advantages and disadvantages to the nation and ourselves of a politically "Solid South," remembering that the political principles which are the heritage of all Americans far transcend in importance loyalty to any political party.

It is not for you or me to write a Declaration of Independence, nor to frame a constitution. It is not for you or me to cross the Delaware nor to be at a Manassas or an Appomattox. It is not for you or me to be a Washington, a Jefferson, a Jackson or a Lincoln. But it is for you and me and all Americans to accept our heritage, given us by great Americans and great events. Yes, we must accept that heritage, love it, appreciate it, work and give for it, that we may say, as did those who signed the Declaration of Independence, "with a firm reliance on the Protection of Divine Providence, we mutually pledge to each other our lives, our fortunes, and our Sacred Honor," that America maintain its greatness and that men shall be forever free. Then, and only then, shall we be able to stand before the men of all nations and sing,

> "My country, 'tis of thee,
> Sweet land of liberty,
> Of thee we sing."

THE BILLROTH I TYPE OF GASTRIC RESECTION

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IN FEBRUARY, 1881, Billroth of Vienna recorded the first success-
ful case of gastric resection and thereby created a new era in
gastric surgery. There had been two previous partial gastrectomies
or pylorectomies, one by Pean in 1879, and the second by Rydigier
of Austria, on Nov. 16, 1880. The technic which Billroth used is
now called the Billroth I and consisted essentially of the removal
of the pyloric portion of the stomach and the anastomosis of the
open end of the duodenum to the open end of the lesser curvature
half of the stomach after closing the greater curvature portion. A
few months later Wölfler, Billroth's assistant, reported 3 more cases
of the same type, but stated that in the future Billroth planned to
close the lesser curvature portion of the stomach and use the greater
curvature part to anastomose to the duodenum. The reason given
was the fear of the formation of a cul-de-sac or diverticulum of the
lower portion of the stomach. This fear has since proved unfounded
but Billroth soon abandoned the procedure in favor of what is
now known as the Billroth II. The reason for this lay in the fact
that leakage occurred in some of his cases at the so-called "deadly
angle" or "fatal angle" where the anastomosis site joined the suture
line of the closed portion of the stomach.

For the most part, gastric surgeons have for many years pre-
ferred the Billroth II or rather some modification of it, such as the
Hoffmeister or the Polya, and the original Billroth I has been to a
large degree discarded. Recently, however, new interest has devel-
oped in this procedure and it is the purpose of this paper to point
out its advantages, limitations and, in general, to encourage its use
in suitable cases. Among its present and recent advocates are Sir
James Walton,[9] Sir Heneage Ogilvie,[6] Sir Grey Turner,[8] A. Garcia
Baron,[1] and several of the Mayo Clinic surgeons.[3,4] It is the method
of choice of Grey Turner, and Walton uses it in nearly all cases of
gastric resection.

For some time it has been known that the susceptibility of the
small intestinal mucosa to ulceration increases as one passes distally
along the small bowel. Harper[2] in 1935 pointed out that dogs which
had been prepared by anastomosing loops of small intestine to
gastric pouches on the one hand, and to skin on the other, developed

ulcers more rapidly when loops of ileum were used to create the fistulae than when jejunal loops were used, and the latter developed ulcers more rapidly than in those cases where duodenal loops were utilized. In his series of ileal loops, perforation of ulcers occurred on an average of 23 days; for jejunal loops the average time was 71 days, and when duodenal loops were used ulcers formed but did not perforate. Whether this same difference in susceptibility is present to the same degree in human patients, and whether upper duodenum is more resistant to ulceration than the same loop a few inches further distal, is not definitely proved but it would seem logical to assume that the same situation might well exist in humans to a greater or lesser degree, and, if so, a short loop of jejunum would be better and safer to use in anastomosing to the stomach than a long loop, and the duodenum would be better than the jejunum at any point. At any rate, Wangensteen and many others prefer a posterior anastomosis rather than an anterior one for this reason and likewise emphasizes the importance of making the proximal loop as short as possible when performing a Hoffmeister anastomosis.

The Billroth I procedure is simpler and can be performed more quickly than a Polya or Hoffmeister in suitable cases because the duodenal stump is not closed. The latter step may be and is often a prolonged and tedious job. Therefore, the Billroth I can be done in poor risk patients with greater safety than other types. There is less danger to the vessels of the transverse mesocolon, there is no suturing of the latter to the stomach around the stoma, and one can dismiss with this procedure the controversy regarding whether to do an anterior or a posterior anastomosis. No one will dispute the fact that it is a more physiologic procedure. The gastric contents pass directly into the duodenum as in the normal subject. Surgeons who have had experience with this method are apparently in agreement that the dumping syndrome is less common although not entirely eliminated, and recently evidence has been found which indicates that there is less loss of fat and protein.[10,11] If the operation is well performed, there appears to be no more gastric retention postoperatively than after other types, although if the stoma is made too small and too much wall is turned in, retention may occur. Furthermore, the decrease in gastric acid is just as great, provided a comparable amount of stomach is removed. It has been my experience that the postoperative convalescence is easier and smoother and patients leave the hospital a little earlier.

There are, however, definite limitations to its use. One must have a duodenum which is mobile or can be mobilized without great difficulty. A stomach which is fixed by inflammatory reaction may

also prevent this technic. Obviously, a duodenum greatly shortened and scarred and buried in adhesions would not be a good case for this procedure. In this respect, however, I have been surprised in some cases of duodenal ulcer with considerable fixation to find that the duodenum can be mobilized rather easily by incising the adhesions transverse to the long axis of the duodenum just beyond the pylorus down to the serosa, inserting the two index fingers and spreading the adhesions apart, then incising the lateral peritoneal reflection on the right side of the duodenum. Another technical step which aids the mobility of the stomach is to divide the left margin of the greater omentum high, or, perhaps more correctly, the lower portion of the gastrosplenic ligament. High ligation of the left gastric and left gastroepiploic vessels also aids in establishing mobility of the stomach. Obese patients are less favorable subjects as a rule than thin ones.

The operation has been criticized on the basis that one cannot remove an adequate amount of stomach and yet bring the stomach and duodenum together without tension. This is not necessarily true. As a matter of fact, total gastrectomies with esophagoduodenostomy have been carried out in certain instances.[5,7] I believe that in most cases three-fourths to seven-eighths of the stomach can be removed and the Billroth I anastomosis completed successfully. Obviously, it would be poor judgment to decide on this procedure in doubtful cases until one actually has resected the stomach and has determined whether or not the stomach and duodenum can be brought together without tension. I have found it useful to leave the clamp on the duodenum until after the resected part is removed and then see how well the duodenum and stomach come together. If a Hoffmeister procedure is decided upon, the duodenal stump can then be closed without having sacrificed anything. I have removed nearly 400 Gm. of stomach and completed the operation as a Billroth I. Figures 1 to 6 show the technic advocated.

There are numerous technics and modifications which have been devised. As stated above, in the original procedure, Billroth closed the portion of the open end of the stomach adjacent to the greater curvature and anastomosed the open end adjacent to the lesser curvature to the open end of the duodenum. Later this was modified and the duodenum was anastomosed to the greater curvature portion of the gastric open end. Haberer used the entire open end of the stomach after decreasing its size by taking "reefing" sutures around the circumference of the mucosa and submucosa. Finney closed the duodenal stump and anastomosed the open end of the stomach to the lateral wall of the duodenum as an end to side procedure. Schoemaker devised a curved and slightly angulated clamp

which removed all the lesser curvature obliquely but joined the greater curvature at a right angle. With modern suturing technics the so-called "fatal angle" should give us little or no trouble. There

Fig. 1. Shows a line of resection. Actually this should show a more radical resection than indicated from the figure.
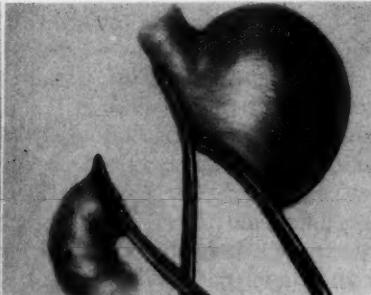
Fig. 2. Shows the application of clamps to the stomach and duodenum and the resected portion of stomach removed.

are two minor points of technic which deserve mention. One is the incising of the anterior duodenal wall longitudinally to create a larger opening (fig. 4) and the other is the use of what virtually amounts to a single row of sutures in making the anastomosis (fig.
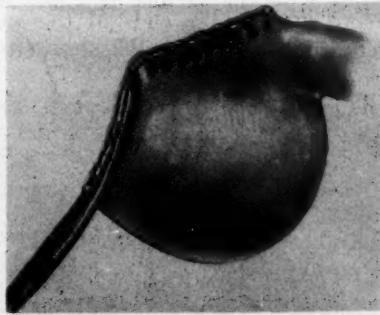

Fig. 3. Shows the lesser curvature part of the stomach closed.

5). This is done by placing through and through interrupted silk mattress sutures as the posterior row inside the lumen of the bowel. Then for the anterior row, interrupted Cushing stitches are placed, using silk. Figure 6 shows completed anastomosis. The interrupted sutures avoid the puckering and gathering that tends to occur with continuous sutures and which tend to decrease the size of the stoma. The Cushing stitch turns in less bowel wall than the Halsted or Lembert and this, too, is an advantage. Anteriorly, a few additional interrupted sutures are placed, incorporating the omentum at the angles. After the anastomosis is completed, one or two

sutures are placed uniting the anterior wall of the stomach to the falciform ligament of the liver. This tends to draw the stomach slightly to the right and thus avoids tension on the suture line. It
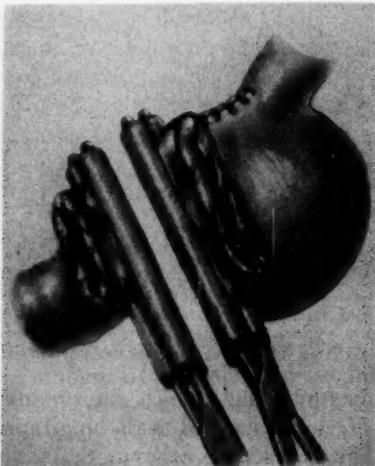

Fig. 4. Shows incision on the anterior wall of the duodenum to enlarge the opening. It also shows the stomach and duodenum being brought together.

should be possible to overlap the ends of the stomach and duodenum about 1 inch or more without difficulty before beginning the


Fig. 5. Shows the anastomosis partially completed, with interrupted mattress through and through sutures on the posterior wall, and interrupted Cushing sutures on the anterior wall.
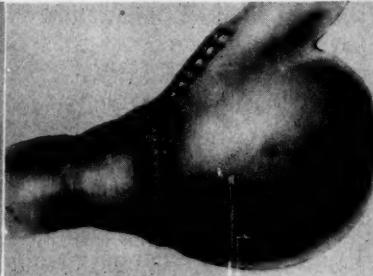
Fig. 6. Shows the completed anastomosis.

anastomosis. The smaller stoma which this procedure provides as compared with the Polya and Hoffmeister procedures is probably a real advantage in preventing the dumping syndrome.

In summary, it is the purpose of this paper to remind the surgical profession of the advantages of the Billroth I procedure in selected

cases. It has numerous advantages over other types of procedures. It is more physiologic. It is easier, quicker and safer. It appears that fat and nitrogen loss is less and the dumping syndrome less frequent. The danger of recurrent ulcer should be less, on theoretical grounds at least, although this has not been definitely proved. Although its field of greatest usefulness is in gastric lesions where the duodenum and stomach are mobile, such as in small gastric ulcers and small gastric carcinomas, it is pointed out that in many cases of duodenal ulcer, large gastric ulcers and large carcinomas with moderate fixation, it can still be carried out with relative ease by performing a few maneuvers to mobilize the duodenum and stomach. It is contraindicated in cases of marked shortening and fixation of the duodenum. It is my impression that the procedure is better tolerated in the average case than other types and that convalescence is quicker and easier.

CASE REPORTS

Illustrating Different Types of Cases in Which Billroth I Method Was Used

CASE 1. Carcinoma of the Stomach. W. E. B., a 48 year old male, complained of cough, dyspnea and stomach distress. His history revealed that he had suffered from asthma since 1941, from a cough and dyspnea on exertion for several months, and from burning distress in abdomen for one month. He reported weight loss of 20 pounds in the past year. He stated that his appetite was poor but he had had no nausea or vomiting; bowels had been regular.

Physical examination disclosed a small, thin man, aged 48, with impaired vision in the right eye. Other findings were: blood pressure 135/95; pulse 80; wheezes and scattered rales, especially in left base; heart, negative except for an occasional dropped beat; abdomen, with slight epigastric tenderness; liver, enlarged to 4 fingerbreadths below the costal margin.

Laboratory findings were as follows: Hematocrit, 60; hemoglobin, 19 Gm. per 100 cc; 10,000 leukocytes per cu. mm.; normal differential. Urinalysis revealed a slight trace of albumin; sedimentation rate, 0; leukocyte count, 2-4 per high power field; block Kahn negative. Bromsulphthalein test was 6 per cent retention in 40 minutes. Sputum was repeatedly negative for tuberculosis. Examination of stools was negative for blood by the guaiac test; plus 1 by benzedine. Roentgenologic examination of gallbladder revealed it to be functioning normally, with no stones. There was a small polypoid lesion on the greater curvature stomach side of the antrum. Gastric analysis revealed no free hydrochloric acid with histamine, highest total acids, 22 degrees; no blood or lactic acid. Electrocardiogram was compatible with mitral stenosis or pulmonale.

Peritoneoscopy was carried out to see if there was any evidence of liver metasis because his general condition was poor. No evidence of metastasis was found.

On Feb. 23, 1951, a subtotal gastrectomy was done with a Billroth I anastomosis, with 80 per cent resection.

The pathologist's report was: infiltrating carcinoma, marked maturity.

The patient was discharged on March 8, 1951, after an uneventful convalescence.

CASE 2. Gastric ulcers. W. S., a 62 year old male Italian miner, complained of abdominal distress. His history revealed that he had suffered for seven years from abdominal aching, dull in character, extending into his back and relieved by taking food. He had had "indigestion" in the same period, with occasional nausea and vomiting. Night distress was frequent and he had taken "Alka-seltzer," "Tums," or milk for relief, especially at night. Because of family troubles, he had been drinking and smoking heavily in recent years. He had been divorced in 1944.

Physical examination revealed a small man, 62 inches tall and weighing 118 pounds. His color was pale and ashen. Other findings were: blood pressure, 145/90; lungs emphysematous; heart negative except for an occasional dropped beat; severe arteriosclerosis; varicose veins bilateral, especially left; slight epigastric tenderness.

Laboratory reports were: hematocrit, 44; hemoglobin 14.2 Gm. per 100 cc.; 4,120,000 erythrocytes per cu. mm.; slight trace of albumin in urine; leukocytes count, 3.5 per high power field; erythrocyte count, 1-3 per high power field; stools, negative for blood. Gastric analysis revealed highest free hydrochloric acid, 24 degrees, total acids up to 33 degrees. Nonprotein nitrogen 32 mg. per 100 cc. Blood Kahn negative. Roentgenogram of stomach revealed two gastric ulcers, one on lesser curvature, quite high, and one on posterior wall.

On Feb. 14, 1951, a subtotal gastric resection with Billroth I anastomosis, with 80 per cent resection done.

The pathologist's report was: peptic ulcer, gastric, nonmalignant; size of resected stomach specimen, 8 by 21 cm.

The patient was discharged on Feb. 24, 1951, after an uneventful convalescence.

CASE 3. Gastric and Duodenal Ulcer. W. B., a 47 year old male farmer, complained of abdominal pain. For 16 years he had suffered from periodic gastric distress relieved by taking food. It had become worse in recent months. He had had several episodes of vomiting. There was no history of melena or hematemesis and no jaundice; bowels were regular. He reported a loss of 10 pounds in weight in recent months.

Physical examination revealed a large male adult aged 47; blood pressure, 110/74. Abdominal findings were negative; a moderate sized nodular was found.

Laboratory reports were as follows: hematocrit, 47; hemoglobin, 16 Gm. per 100 cc.; 7,400 leukocytes per cu. mm.; normal differential; blood Kahn negative; negative urinalysis; nonprotein nitrogen, 34 mg. per 100 cc. Basal metabolic rate, plus 27. Gastric analysis: highest total acids, 40 degrees; highest free hydrochloric acid, 20 degrees, after histamine, no blood.

Roentgenogram revealed a gastric ulcer on lesser curvature 1 cm. in diameter. Duodenal bulb deformed and contracted 30 per cent, with 3 hour gastric retention. Nightly aspiration showed about 400-500 cc. retention, which later was 180 cc.

On Feb. 19, 1951, a subtotal thyroidectomy was done and on March 7, 1951, a subtotal gastric resection with a Billroth I anastomosis.

Pathologic report was: benign peptic ulcers, gastric and duodenal; weight of resection specimen, 350 Gm.

Convalescence was uneventful and the patient was discharged on March 14, 1951.

CASE 4. Duodenal Ulcer. Mrs. T. A. J., aged 47, had suffered from intermittent pain in stomach for many years. She had had periodic vomiting with relief. She reported that milk relieved her for one hour and that she had also taken soda and some "powders." There was intolerance to certain foods. She reported some weight loss recently.

Physical examination revealed a woman aged 47, height 62½ inches, weight 117 pounds. Other findings were: blood pressure 120/80; pulse, 90; rather poor dental hygiene; small adenomatous goiter; heart, rapid but negative; lungs, clear; breasts, negative; abdomen, moderate epigastric tenderness. Pelvic examination: relaxed perineum and vaginal walls; badly lacerated cervix.

Laboratory reports were: gastric acids, total, 39 degrees, free hydrochloric acid, 30 degrees; hematocrit, 42.5; 6,600 leukocytes per cu. mm.; sedimentation rate, 8; urine was negative; blood Kahn negative; basal metabolic rate, minus 7.

Roentgenologic examination revealed a duodenal ulcer with 70 per cent, 3 hour retention.

On January 31, 1951, gastric resection with Billroth I anastomosis, 80 per cent resection, was done. Convalescence was uneventful.

Pathologic report was: duodenal ulcer; specimen 12 by 24 cm.

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THE SURGICAL TREATMENT OF PANCREATITIS

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THE pancreas is a flattened elongated organ lying on the posterior wall of the abdomen at the level of the first or second lumbar vertebra. It lies posterior to the stomach and forms a large part of the posterior wall of the lesser peritoneal sac. The head of the gland lies in the duodenal loop. The uncinate process extends from the head of the gland posterior to the portal vein and the superior mesenteric artery. The tail of the gland extends to the left, well up into the hilus of the spleen. The duct of Wirsung runs the length of the gland and empties into the duodenum at the ampulla of Vater as a common channel with the bile duct in about 85 to 90 per cent of the cases. The accessory duct of Santorini, which drains a portion of the head of the gland, empties separately into the duodenum about 2 cm. above the papilla.

The gland is surrounded by large blood vessels: The pancreaticoduodenal artery encircles the head of the gland; the splenic and gastrohepatic arteries lie superior to it; and the portal vein and superior mesenteric artery are on the posterior side. The gland itself is supplied with a rich network of small blood vessels which bleed briskly after trauma to the organ.

Histologically the pancreas is composed of (1) the acinar cells, (2) the excretory ductal system, (3) the islands of Langerhans, and (4) the interlobular connective tissue.

The acinar cells are responsible for the external secretion of the gland. This secretion, which measures about 700 cc. daily, is a clear, slightly mucoid liquid resembling saliva and contains a number of important digestive enzymes. It is always distinctly alkaline, but its composition changes from time to time depending upon the nature of the secretory stimulus. The secretion seems to be continuous and increases during periods of digestion. The protein content of the secretion is such that it is possible for a patient to lose

as much as 6 Gm. of protein daily from an external pancreatic fistula.

The protein fraction of the pancreatic juice contains the digestive enzymes. These may be grouped into three classes: the proteolytic, which digests protein; the amylolytic, which digests starch; and the lipolytic, which digests fat. Thus the external pancreatic secretion has the ability to digest all three classes of foodstuffs.

Proteolytic Enzymes. It was originally thought that the proteolytic power of pancreatic juice was derived from one enzyme—trypsin. Actually six different enzymes of this type have been isolated, and others are known to be present. The two most important are trypsin or chymotrypsin. In most instances, trypsin is present in pure pancreatic juice in an active form which has been called trypsinogen. A hormone called enterokinase, which is formed in the duodenal mucosa, activates the trypsinogen to trypsin.

Rich and Duff[1] have pointed out that under certain conditions, such as a recent heavy protein meal, the pure pancreatic juice may contain a significant amount of active trypsin.

Amylolytic Enzymes. Amylase is the most important of the starch splitting enzymes. It occurs in the blood under normal conditions but is greatly increased during acute inflammation of the pancreas. The cause of this increase is not definitely known. It has been suggested that damaged acinar cells allow the escape of the enzyme into the blood stream.

Lipolytic Enzymes. Pancreatic lipase is a powerful fat splitting enzyme which bears the burden of fat digestion. It normally works in close cooperation with bile, and the combination digests fat four or five times faster than lipase alone.

There are three sources of stimulation for external pancreatic secretion. They are:

A. Nervous—The nervous stimuli are from the parasympathetic system and are mediated almost entirely via the vagus. The sympathetic nerves have little effect on the secretion other than through their control of the blood flow to the gland.

B. Hormonal—When hydrochloric acid from the stomach passes into the duodenum, a hormone is produced in the duodenal mucosa. This hormone is called secretin and it acts directly on the acinar cells of the pancreas to elicit a profuse watery secretion which is poor in ferments.

C. Secretogogues—A diverse group of substances, such as protein derivatives, fats, fatty acids, and water when introduced into

the duodenum may elicit an increase in pancreatic secretion rich in ferments. The exact mechanism of stimulation is in doubt, but it seems certain that these agents act independently of secretin.

LESIONS OF THE PANCREAS

Acute Pancreatitis. It seems evident that there are several different conditions that should be considered etiologic agents in the production of acute pancreatitis. Obstructions at or near the ampulla of Vater in the major duct either by stones, spasm of the sphincter of Oddi, or tumors are at times associated with acute pancreatitis. Such obstructions may be either so situated that reflux of bile from the common duct into the pancreatic duct is encouraged, or the blockage may obstruct the pancreatic duct in such a manner that bile reflux is prevented. Trauma and ductal metaplasia may also be associated with acute inflammation. The frequent history of excessive alcoholic intake in these patients has been mentioned by many observers. Not only is the disease frequently observed in the patient with chronic alcoholism, but also attacks of acute pancreatitis are frequently associated with bouts of excessive alcoholic intake. It has been suggested that an alcoholic duodenitis may elicit additional spasm or actual stricture of the sphincter of Oddi and thereby be an important etiologic factor in the disease. Actual proof of such a mechanism is lacking. It is possible that alcohol exerts its influence on the acute phase of the disease by acting as a secretagogue and stimulating markedly the secretory activity of the gland.

Despite several known causes of acute pancreatitis, the exact etiological mechanism remains obscure in the majority of the cases.

CHART 1

Patients Admitted to the Veterans Administration Center, Wadsworth Hospital, with a Diagnosis of Acute Pancreatitis

Year	No.
1947	2
1948	10
1949	19
1950	30
1951	1 (Jan.)

During the past four years 62 patients have been admitted to the Veterans Administration Center, Wadsworth Hospital, suffering from an attack of acute pancreatitis. With the advent of the use of the amylase test as a diagnostic tool, the number of cases diag-

nosed has increased rapidly during the last few years. This increase is shown in chart 1.

In other reported series the disease has been most frequently encountered in patients between the ages of 40 years to 70 years. However, in this series there was an even distribution of age from 20 years to 60 years, and 3 of the patients were over the age of 70 years.

The acute edematous or interstitial type of pancreatitis has been frequently diagnosed by means of clinical findings and laboratory tests and accounts for 92 per cent of the cases seen in this hospital. Acute hemorrhagic pancreatitis has been seen much less frequently and has been attended by a very high mortality rate.

CHART 2

Temperature Range

Temp.	No. Cases	Per Cent
98.6° or less	34	55
98.6° to 100.6°	20	32
100.6° to 101.6°	6	10

Symptoms: Pain is the most prominent single symptom of the disease and typically starts suddenly in the upper abdominal region and radiates to the back or flank. The severity of the pain is a rough index of the degree of pancreatic damage. The onset of the pain was sudden in 40 of our cases and gradual in 20. There was no complaint of abdominal pain in 2 cases. The pain radiated to either the back, the chest, or the shoulder in approximately half of the cases. Nausea and vomiting promptly followed the pain in 70 to 75 per cent of the cases and was frequently severe enough to be one of the most prominent symptoms.

Fever was mild or absent (chart 2).

Shock occurs rarely but is an ominous sign and is usually indicative of severe pancreatic damage of the hemorrhagic type. In fact, 42 per cent of the cases in this series showed elevation of the blood pressure. If a mild case is inadequately treated, late shock (24 hours or more after onset) may develop from persistent pain, vomiting, and dehydration.

Other significant physical findings are usually centered about the abdomen. Tenderness is almost always present. Rigidity and rebound tenderness are often found. Distention, voluntary guarding, and diminished peristalsis may be present.

Discoloration of the flanks (Turner's sign) or the umbilicus

(Cullen's sign) indicate extravasation of blood into the retroperi-
toneal tissues. This rarely occurs but when present indicates serious
destruction of the gland. Turner's sign, which usually develops on
the second or third day after an attack, is almost pathognomonic
of acute hemorrhagic pancreatitis.

A leukocytosis with a white blood cell count from 10,000 to 20,-
000 polymorphonuclear leukocytes per cu. mm. was present in over
half of our cases.

The serum amylase level, the most important single diagnostic
test, was elevated in all but 7 of the cases.

Sugar was present in the urine specimen at admission in 18 per
cent of the cases. Of the 17 cases tested, the blood sugar level in 5
of them during the fasting state was over 120 mgm. per cent.
Glucose tolerance curves on 8 patients revealed 2 cases with a
diabetic type of curve.

Siler and Wulsin[2] summarize a discussion of the relationship of
diabetes to pancreatitis as follows: "The pancreas can sustain much
damage without the advent of diabetes, but in all diseases of that
organ one must look for the presence of diabetes, and be aware that
it may develop in the future if there is further involvement of the
pancreas. If it does develop, it must be handled and treated as
diabetes in its usual form. Once developed it tends to remain mild
or only moderately severe."

A segmental ileus which usually involved the jejunum was present
by roentgenologic examination in 65 per cent of our cases. Chole-
cystography in 41 cases demonstrated a malfunctioning gallbladder
in 19 (46 per cent) of the patients.

Differential Diagnosis: The diagnosis of acute pancreatitis has
been greatly facilitated by the use of the serum amylase test. There
are, however, other acute abdominal conditions in which a moderate
elevation of the serum amylase occurs. The fact that serum amylase
returns to normal levels rather promptly after the early stages of
the disease must also be borne in mind. During a mild attack, the
serum amylase level may remain elevated for only a few hours.

Differential diagnosis is especially important in cases which in-
volve those conditions in which delay in operation may invite serous
complications, such as perforated peptic ulcer or acute appendicitis.

The conditions most frequently considered in the admitting and
differential diagnoses were in order of frequency as follows: acute
cholecystitis, perforated ulcer, penetrating ulcer, duodenal ulcer
without perforation, acute appendicitis, intestinal obstruction, and

mesenteric vascular occlusion. The supradiaphragmatic conditions most frequently considered were pneumonia and coronary occlusion.

Treatment: If the correct diagnosis can be made in a case of acute edematous pancreatitis, the treatment is nonoperative. Operation has been advised (1) if a positive diagnosis could not be established, (2) if acute hemorrhagic pancreatitis were strongly suspected, or (3) if it were a case of recurring or relapsing pancreatitis. If the diagnosis has been uncertain and the presence of acute pancreatitis is determined at operation, no further procedures are performed. The abdomen is closed without drainage.

The nonoperative therapy is directed toward the symptomatic treatment of the pain and nausea and toward diminishing the secretory activity of the gland. Demerol has been used to control pain. Morphine is avoided as it elevates the pressure in the common duct and may thereby aggravate the disease process by encouraging the reflux bile into the pancreatic duct. A nasal-gastric tube is passed, and constant suction is maintained to remove gastric secretions and diminish the production of secretin. Atropine and, more recently, Banthine have been administered to block vagus secretory impulses and to relax the sphincter of Oddi. The reduction of acid gastric secretion by these drugs probably also assists in reducing pancreatic secretory activity.

An antibiotic, usually penicillin, is administered routinely to increase resistance to infection in the event that a portion of the gland should later become gangrenous or an associated infectious cholangitis develop.

Adequate hydration is maintained by the administration of intravenous fluids.

Treatment in the cases of acute hemorrhagic pancreatitis has been less standardized. In general, treatment has been the same as the nonoperative treatment of the cases described above except that additional vigorous therapy is given to combat shock. If the patient's general condition can be sufficiently improved, the lesser peritoneal sac is drained at operation, and any obviously gangrenous pancreatic tissue is removed.

Chronic Pancreatitis. It has been estimated that approximately one-third of the patients who have one attack of acute pancreatitis will eventually have additional bouts of the disease. Chronic inflammation, scarring, and ultimately atrophy of the gland develop if recurrent attacks continue. At the onset of the disease it is not possible to predict which patients will be the ones in whom the chronic form of pancreatitis will develop.

Chronic pancreatitis is most commonly found in patients who are in the third or fourth decade of life. There is frequently a concurrent history of chronic alcoholism.

Symptoms: Intermittent attacks of pain become more frequent until a constant pain in either the epigastric region or in the back develops and is the cardinal symptom of the disease. Clinical evidence of disturbances of the external pancreatic secretion is usually ill defined. Steatorrhea is not common, but dyspepsia, anorexia, and diarrhea are frequently seen. Interference with the function of neighboring structures, which is due to scarring and fibrosis of the organ, may produce important clinical symptoms—for example, biliary obstruction resulting from a constriction of the intrapancreatic portion of the common duct.

Calcification frequently develops in the chronically diseased pancreas and may take one of two forms: (1) one or more relatively large calculi may lodge in the major ducts; this condition is spoken of as pancreatic lithiasis, or (2) diffuse calcification of the pancreas may be present and indicates numerous minute calcified areas in the finer radicles of the ductal system. The origin of such calcification is not understood. The stones contain calcium and are radiopaque. If removed surgically they frequently reform. Once present they promote further damage to the gland by preventing proper drainage and causing dilatation of the ductal system and atrophy of the secreting parenchyma.

Treatment: The treatment of chronic pancreatitis presents a difficult problem and results may be unsatisfactory regardless of the type of therapy used.

Nonsurgical therapy involves the control of pain, dyspepsia, and pancreatic insufficiency. Addiction to morphine or other analgesic drugs is common but can usually be corrected if the constant pain can be alleviated.

A variety of surgical procedures has been proposed for the treatment of chronic pancreatitis and its complications. In general, these may be grouped under the following classifications:

1. Operations on the biliary tract
    (a) cholecystectomy
    (b) exploration and prolonged drainage of the common duct

2. Control of pain by division of sensory nerves
    (a) splanchnicectomy
    (b) celiac ganglionectomy

3. Prevention of biliary reflux
    (a) division of the sphincter of Oddi
    (b) division and reimplantation of the common duct or pancreatic duct

4. Methods to inhibit pancreatic secretion
   (a) subtotal gastric resection
   (b) vagus nerve resection
   (c) ligation of the duct of Wirsung

5. Excision of gland
   (a) partial pancreatectomy
   (b) total pancreatectomy

Operations on the biliary tract: Surgical exploration should be carried out in any case of chronic pancreatitis in which there is disease of the biliary system. In most instances cholecystectomy with exploration and prolonged T tube drainage of the common duct will be indicated. In the series of cases of pancreatitis at the Wadsworth Hospital, 13 patients who had abnormal cholecystograms were operated on. In 12 of these, cholecystitis, cholelithiasis, or choledocholithiasis were found either singly or in combination.

Our experience with a small number of cases would indicate that elimination of disease of the biliary system would bring about improvement in approximately 50 per cent of the cases of early chronic or relapsing pancreatitis.

Control of the pain of chronic pancreatitis by division of the sensory autonomic nerve fibers has been reported by a number of authors.[3,4,5] This procedure has usually entailed division of the splanchnic nerves and interruption of the sympathetic chain.

Temporary relief of pain by excision of the celiac ganglia has been reported by Grimson, Hesser, and Kitchin.[6]

Alleviation of pain following sympathetic denervation seems to have been routinely accomplished in the several rather small series of cases which have been reported. However, objection has been raised to denervating the abdominal viscera. The procedure has also been questioned because steatorrhea, dyspepsia, and weight loss may continue despite the relief of pain.

Other methods of treatment deserve evaluation. However, for the relief of the cardinal symptom of the disease—pain—splanchnicectomy remains an effective procedure.

Prevention of biliary reflux: Doubilet and Mulholland[7] have demonstrated the importance of spasm of the sphincter of Oddi accompanied by reflux bile through a common channel at the ampulla into the pancreatic duct as a causative factor in pancreatitis. They have obtained relief of symptoms in a large number of patients with chronic pancreatitis by dividing the sphincter of Oddi. Even in cases of pancreatic calcification,[8] pain has been abolished, recur-

rent attacks of severe pancreatitis prevented, and the progress of the disease arrested by this procedure.

We have performed transduodenal sphincterotomy in 8 cases of chronic pancreatitis. In 3 cases the procedure has been effective for a relatively short follow-up period. Four patients have continued to have severe symptoms and have required further therapy. The other patient eventually succumbed to an extensive gangrenous pancreatitis.

Sphincterotomy undoubtedly offers relief for certain patients with chronic pancreatitis; others, however, are not benefited. The duration of the disease may be important in determining the response to sphincterotomy. At present, the criteria for selection of patients for operation have not been established.

Bowers[9] believes that diversion of the flow of bile from the ampulla of Vater can best be accomplished by dividing the common duct and implanting it into a Roux-Y jejunal limb. He expresses the hope that the results of this procedure will be more permanent than sphincterotomy. Hay has attempted to achieve the same result by reimplanting the severed duct of Wirsung into the duodenum away from the ampulla. The hazards of stricture at the site of duct-intestinal anastomosis dictates extreme caution in the selection of these procedures.

Methods to inhibit pancreatic secretion: Richman, Colp, and Lester[10] state in a preliminary report, "Subtotal gastrectomy reduces gastric acidity and diverts the flow of chyme from the duodenum and upper jejunum into that portion of the small intestine in which secretin is found in diminishing quantities. These two factors may be considered to be responsible for diminished secretin output and, thus, lessened stimulation of pancreatic secretion. In addition, the diversion of the food current from the papilla of Vater reduces irritation and spasm of the sphincter of Oddi." They report encouraging results in 7 patients treated by this procedure but emphasize that the method is not advocated for routine use until further experience has been obtained.

McCleery, Kesterson, and Schaffarzick[11] report vagotomy in 11 cases with definite improvement in all. They emphasize the importance of abolishing psychogenic stimuli mediated via the vagus nerve that affect the ampullary muscle and the pancreatic and gastric secretion. Seven of the 11 patients had been followed between one and two years. The permanency of the effect of vagotomy has, however, been questioned. Further experience with the method is indicated.

Ligation of the duct of Wirsung might be expected to result in

acinar atrophy and possibly cessation of certain of the symptoms of pancreatitis. Other methods which give symptomatic relief without completely abolishing the secretory activity of the gland would be desirable.

Excision of the gland: Restriction of the disease process to the distal portion of the gland as demonstrated by localized areas of calcification may be treated by partial pancreatectomy. Early improvement may be expected; however, involvement of the remaining portion of the gland may occur. Total pancreatectomy may be considered if the entire gland is involved and if more conservative methods have failed. Dennis[12] has emphasized the technical difficulties which may be encountered in this operation. Permanent replacement therapy of both the internal and external pancreatic secretions are required postoperatively.

As previously stated, the treatment of chronic pancreatitis poses a difficult problem. Results are frequently unsatisfactory regardless of the method or methods of therapy selected. The following methods of treatment are listed in the order in which they are employed, if necessary: (1) treatment of biliary tract disease if it is present; (2) transduodenal sphincterotomy; (3) vagotomy combined with minimal subtotal gastric resection; (4) splanchnicectomy; and (5) if there is evidence of limited disease, partial pancreatectomy.

SUMMARY

A brief summary of the anatomic and physiologic characteristics of the pancreas as related to pancreatitis has been given.

A review of a series of cases of acute pancreatitis and the results of treatment are presented.

The various methods of therapy recommended for chronic pancreatitis are discussed.

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THE DIAGNOSIS OF THROMBO-EMBOLIC DISEASE

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THE diagnosis of thrombo-embolic disorders ordinarily presents no difficulty though at times puzzling exceptions are encountered. Their prompt recognition and treatment are dependent upon an awareness of their possible occurrence and familiarity with the typical clinical picture which characterizes them, as well as with the variations from the usual findings which sometimes occur. For convenience of presentation I shall divide my discussion into five parts —first, a consideration of superficial thrombophlebitis; second, thrombophlebitis of deep veins; third, phlebothrombosis or bland thrombosis; fourth, pulmonary embolism; and fifth, septic thrombophlebitis.

Superficial thrombophlebitis is generally recognized with ease. The veins most commonly affected are segments of the major and lesser saphenous veins, the cephalic and the median basilic veins. Not infrequently thrombophlebitis may develop in superficial varicosities. The disorder may appear after trauma or intravenous injections or infusions, but it may also develop without any apparent cause. The patient complains of localized pain and tenderness. At times the pain may be mild, at other times very severe. Upon examination the affected segment of the vain may be palpated as a firm tender cord or there may be an overlying elongated ovoid area of cellulitis which prevents definite palpation of the occluded vein itself. There may be no edema at all or there may be minimal or moderate edema of the tissues distal to the site of venous involvement. The limb as a whole is not swollen and, if significant edema is present, one is likely to be dealing with associated deep venous thrombosis. In some cases the hand or foot shows obvious evidence of vasoconstriction with coolness, pallor and perhaps hyperhidrosis. The condition is sometimes confused with ordinary cellulitis, but the characteristic distribution of the area of cellulitis in cases of thrombophlebitis, overlying as it does the course of a vein and almost always being an elongated ovoid in shape, usually permits easy differentiation. The condition is recognized with greater difficulty when a very small area of a vein is involved. In such circumstances one must naturally consider other possibilities such as ery-

thema nodosum, erythema induration, nodular syphilids and tuberculids and nonspecific panniculitis. When such short segments of veins are affected, the process is often a migratory affair recurring from time to time and involving successively different areas. In cases of this sort it is obviously of utmost importance to establish or rule out the presence of thromboangiitis obliterans. Some individuals have recurrent attacks of thrombophlebitis involving segments of the major saphenous vein without migration to other areas. They are less likely to have diffuse arterial and venous inflammatory disease as an underlying cause.

The diagnosis of deep thrombophlebitis also ordinarily presents no real problem. The patient complains of pain in the region of the affected vein and of swelling, and generally of a diffuse aching of the limb. Not infrequently there is associated general malaise. The swelling of the limb is variable in degree and extent and is dependent upon the location of the thrombophlebitic process and the limitation or diffuseness of involvement of collateral venous pathways. In iliofemoral thrombophlebitis the entire extremity is apt to be markedly swollen up to the groin. In femoral thrombophlebitis the swelling generally extends proximally only to the knee area or the lower portion of the thigh. Popliteal occlusion ordinarily results in edema of the distal part of the leg, the foot and ankle. Edema is less extensive or entirely absent when the deep veins of the calf are affected and, when present, primarily involves the foot and ankle, though sometimes the lower portion of the leg is also enlarged. The entire upper extremity is likely to be swollen in cases of subclavian thrombophlebitis, while swelling may be more sharply limited to the forearm and hand in cases of axillary or brachial occlusion. In almost all instances of thrombophlebitis there is marked or moderate tenderness on deep palpation over the affected segment. Sometimes there is evidence of cellulitis in this area. The affected limb generally shows increased warmth, though there may be associated vasospasm of the foot or hand. A mild elevation of temperature, pulse rate and leukocyte count may be present and occasionally high fever. The absence of fever, tachycardia and leukocytosis does not, however, rule out the presence of active thrombophlebitis. Because of the characteristic pain, tenderness over the affected vein and diffuse swelling of the limb, deep thrombophlebitis is rarely mistaken for any other condition.

On rare occasions thrombophlebitis may be confused with sudden arterial obstruction, either embolic or thrombotic. These are the cases of phlegmasia cerulea dolens. The patient may have an abrupt onset of massive swelling of the lower extremity, intense diffuse deep pain, and a violaceous discoloration of the foot and leg

and sometimes of the thigh. The foot rapidly becomes cold and generally numbness and paresis develop. The pulses may become less forceful and in rare circumstances disappear from the foot and ankle. Recovery may take place, or gangrene may ensue. In other instances this train of events may be superimposed upon the usual picture of iliofemoral thromboph!ebitis. The obvious associated arterial circulatory insufficiency may make one consider a primary arterial disorder. The massive swelling and marked cyanosis, however, generally differentiate the condition readily from arterial embolism or thrombosis. Any edema which develops in these cases is likely to be minimal or moderate and to follow rather than precede signs of arterial obstruction. In unusual circumstances sudden arterial occlusion may be followed by massive venous thrombosis and cases of this kind might more easily be confused with primary phlegmasia cerulea dolens. If they are observed from the first or a clear history is obtainable, they are distinguishable by virtue of the development of ischemia before the onset of swelling and cyanosis.

Much greater difficulty is encountered in the recognition of phlebothrombosis or quiet, bland thrombosis. Here there is absence or only suggestive evidence of the signs of inflammatory reaction in the involved vein characteristic of thrombophlebitis. The patient, indeed, may have no complaints at all and the condition will be recognized only by frequent and careful physical examination. The commonest sites of involvement are the deep veins of the calf and, less often, the deep veins of the heel and along the plantar surface of the foot. In the latter instances the only sign may be tenderness on deep palpation. If the patient is ambulatory he may notice discomfort on weight bearing. Many patients with phlebothrombosis of the deep veins of the calf are completely unaware of its presence. Some complain of a deep mild calf discomfort. There may or may not be a slight tachycardia and mild elevation of temperature. The most reliable sign pointing to the presence of this condition is tenderness on deep palpation of the calf muscles. This is elicited by making gentle but deep pressure over the posterior aspect of the calf or by deep compression of the calf muscles from the mesial and lateral aspects. Since minimal pain may be caused by very deep pressure in normal individuals it is important to compare the two extremities. The calf pain which occurs as a response to forcible dorsification of the foot as described by Homans is a valuable confirmatory sign. One may be misled, however, in certain instances. Pain is apt to occur upon dorsification of the foot whenever any significant shortening of the tendo Achilles is present. The same is true in cases of paresis of the foot extensors as sometimes occurs in

patients long bedridden, presumably from the continued downward pull of bed clothes upon the foot. In rare cases there may be slight swelling of the leg, ankle and foot. In others no distinct swelling may be evident on inspection but may be clear from prior daily measurements of calf circumference. The calf is generally no warmer than the contralateral calf and vasospasm of the homolateral foot is much less common than in cases of thrombophlebitis. Not infrequently, in spite of the paucity of signs and symptoms, the evidence of phlebothrombosis is convincing. In many cases, however, the evidence is only suggestive. In them it is wise to proceed on the assumption that the suspicion is well founded.

Similar quiet thrombosis without significant evidence of inflammatory reaction may occur in other vessels such as the femoral and axillary veins. In cases of this sort, swelling of the extremity occurs without significant pain or tenderness. To be sure, after a period of time a mild sense of aching may be felt in the limb and an uncomfortable tightness. If the condition develops while the patient is under observation, it is generally recognized. The presence of the swollen limb and perhaps minimal tenderness on deep pressure over the affected vein are sufficient for the presumptive diagnosis of deep venous thrombosis. If there is evidence pointing to the presence of a neoplastic mass which may be compressing an important adjacent vein, it is difficult to be sure whether venous thrombosis has, or has not, taken place. This is not infrequently the case in instances of iliac and inferior or superior vena caval obstruction. Where a mass is clearly present, it points to compression from without rather than to thrombotic occlusion within the vein as the causative factor. Not infrequently such cases of bland thrombosis occur while the patient is in good health and ambulatory, most commonly in the iliac, femoral or axillary veins. The latter cases may be preceded by some unusual muscular effort. They demand careful examination in order to exclude venous obstruction from one of the shoulder compression syndromes. They can ordinarily be recognized because of the predominance of signs and symptoms of venous occlusion without significant evidence of arterial circulatory impairment or the neurogenic manifestations so common in cases of scalenus anticus syndrome, cervical rib, the costoclavicular compression syndrome, hypertrophy of the subclavius muscle, and the hyperabduction syndrome, as well as by absence of the specific signs suggestive of these conditions. The person who has developed relatively painless swelling of the lower extremity, made worse by dependency and relieved by elevation, is likely to have had a deep venous thrombosis and often venography or the subsequent appearance in neglected cases of dermatitis, pigmentation or ulceration may offer confirmatory

evidence. On occasion, however, one meets with such cases in which there is no evidence, venographic or otherwise, of deep venous involvement and in which the proper diagnosis remains obscure. The recognition of bland venous thrombosis in patients treated by plaster cast for traumatic injury of an extremity is particularly difficult since swelling may go unrecognized or may be thought consistent with the reaction to the trauma alone. Often the occurrence of venous thrombosis is recognized only after the cast has been discarded by the development of persistent symptoms and signs of venous occlusion. Undoubtedly the diagnosis of deep thrombosis in such cases will always be difficult. Whenever unusual edema develops in these cases, however, one should be suspicious of thrombosis.

When a patient presents himself with swelling of a lower extremity without history of the characteristic story of thrombophlebitis, a number of conditions must be considered. In rare cases edema may be the result of incompetency of the valves of the femoral venous system. This diagnosis is difficult to establish. My colleagues and I are encouraged by our experiences with functional venography in which both dependency and the pumping action of muscular activity are utilized, but we feel that continued study is necessary before the final value and limitations of this technic are evident. More easily recognized is a poorly understood syndrome characterized by mild or moderate edema, a tendency to cyanosis and vasospasm of the feet, a tendency for increase in symptoms at the time of the menses and during warm weather, and a proneness to occur in young women. The edema characteristic of cardiac or renal failure rarely presents any difficulty in recognition. More often confused with venous thrombosis is the edema which is prone to develop in patients who have arterial circulatory deficiency and, generally because of pain in the recumbent position, sit for long periods of time with the extremity dependent and still and who take little active exercise because of discomfort on walking. One of the most important conditions to differentiate from the edema resulting from venous thrombosis, and unfortunately one often confused with it, is post-traumatic reflex edema. It is essential that the two be differentiated from one another since the proper early management of the two conditions differs widely. In reflex edema there is always the history of an injury, though the trauma is often mild. The edema is usually limited to the foot and distal portion of the leg or to the hand and distal portion of the forearm, though in neglected cases it may become more massive and extend up to the knee or elbow. Though not invariable there is usually present some discomfort made worse by weight bearing or active use of the part. There is always present some muscular paresis and quite often

hypesthesia. Some vasomotor alteration invariably accompanies the condition. Usually at first there is vasoconstriction with coldness, pallor, commonly hyperhidrosis, and not infrequently cyanosis. Hyperemia may rapidly replace the vasospasm, though if untreated the hand or foot generally exhibits sympathetic overactivity again after a period of time. If one is aware of this syndrome, it should be recognized without trouble. Chronic lymphedema or elephantasis is occasionally mistaken for the edema of venous occlusion. The hard, brawny character of the swelling and the slowness with which it subsides upon elevation of the part should establish the correct diagnosis with ease. The subcutaneous trabeculation seen on soft tissue roentgenography is pathognomonic.

The diagnosis of pulmonary embolism is relatively easy if all the typical findings are present, sudden onset of chest pain and dyspnea, cough with bloody expectoration, circulatory collapse, cyanosis, physical and radiologic signs of pulmonary infarction and localized pleuritic response. Rather typical cases may, however, be confused at times with such vascular catastrophes as acute coronary occlusion. Unfortunately, the clinical picture is variable and sometimes there is only chest pain and suggestive changes in the lungs. Though the diagnosis may be uncertain one must consider such cases presumably to be examples of pulmonary embolism until this diagnosis can be established or ruled out. If a patient has suggestive respiratory signs and symptoms and also evidence of peripheral venous thrombosis the problem is made easier. The absence of obvious evidence of venous thrombosis necessitates a careful survey in an effort to recognize the more occult cases. In some, one cannot establish the source of embolism. In cases of doubt, the existence of thrombosis must be assumed and the patient treated accordingly.

The diagnosis of septic thrombophlebitis is easily established when peripheral thrombophlebitis is evident and the patient has the septic fever and positive blood cultures characteristic of bacteremia. Though the diagnosis is more difficult to establish, one should certainly consider it as a possibility in cases of bacteremia without apparent peripheral thrombophlebitis. A history of recently induced abortion or other intrauterine instrumentation in such cases is highly suggestive of pelvic thrombophlebitis. The association of chills and fever with acute appendicitis should make one strongly suspicious of associated pyelophlebitis. All too often it is recognized only later when jaundice develops and points to the presence of liver abscesses.

I have attempted in this discussion to point out the salient points in the differential diagnosis of thromboembolic disorders. The wis-

dom of separating cases of deep venous thrombosis into those with thrombophlebitis and those with phlebothrombosis is challenged by some. It is undoubtedly true that the distinction is at times tenuous and that often relatively quiet thrombosis, without significant inflammatory reaction and typical thrombophlebitis, occur simultaneously. It is also true that cases of phlebothrombosis sometimes exhibit later classical signs of thrombophlebitis, especially if the best treatment has not been carried out. I have felt that the clinical differentiation, when possible, was worthwhile, however, not only because the hazard of embolism is greater in phlebothrombosis but also because the early recognition of these cases is difficult and is dependent upon a constant awareness of its possible occurrence. In all varieties of thromboembolic disease prompt diagnosis and early effective and continued treatment is necessary not only to insure the patient maximal safety but also to decrease the hazard of annoying and often disabling sequelae.

MODERN CONCEPTS OF INTESTINAL ANTISEPSIS

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O UR present-day concepts of intestinal antisepsis have arisen within approximately the past 10 years. During the preceding 75 years attempts had been made to sterilize the gastrointestinal tract of animals and man without success. There are a large number of misconceptions about the bacterial flora of man and the manner in which it develops from infancy. It was long thought that the bacterial flora in the infant was very much different from that of the adult, and this variation was considered to be due to the fact that the infant was taking milk. Some recent studies in this institution, by Dr. Robert Wise, demonstrated a very rapid establishment within two or three days of essentially the same bacterial flora in the gastrointestinal tract of the infant as that of adult man, regardless of whether the child was breast- or bottle-fed. In either event the diet was primarily milk. It has also been demonstrated in the case of man that the diet of the adult has little effect either on the qualitative or the quantitative bacterial population. However, there are certain animal species, especially among the rodents, in which the bacterial flora can be very markedly affected by the type of diet. If the diet is largely protein there will be a putrefactive type of organisms, while if the diet consists of carbohydrates only, the bacteria are predominantly nonputrefying organisms.

An antibacterial agent to serve as a practical intestinal antiseptic should probably have the following properties:

1. A low toxicity for the patient.
2. A broad bacterial spectrum.
3. Not destroyed by digestive ferments.
4. Prevention of the development or overgrowth of resistant bacterial variants.
5. Rapidity of activity.
6. Limited absorption from the gastrointestinal tract.
7. Action in the presence of foods and other foreign substances, thereby permitting adequate intake of food and fluids.
8. Be of value in the mechanical cleansing of the bowel without causing dehydration.
9. Not cause irritation of the gastrointestinal tract.

10. Not interfere with tissue growth and repair.
11. Low dosage.
12. Solubility in water.
13. Palpability. ⌐
14. Will inhibit the excessive growth of fungi.

Our experience would indicate that it is most important that the substance be poorly absorbed from the gastrointestinal tract, because by poor absorption the host is not subjected to large amounts of the drug in contact with its own body tissue. It has been found that, whenever the compound is freely absorbed from the gastrointestinal tract, although it is relatively nontoxic, untoward toxic reactions occur frequently enough to preclude its use. The ideal would be the use of a substance in the gastrointestinal tract which is not absorbed and would remain essentially outside of the body of the host without subjecting the patient to the dangers of direct cellular damage by these potent chemicals or antibiotic agents.

As our experience has grown in this field we have come to recognize that practically all of the studies on intestinal antiseptics must be made on man, because it is impossible to carry from one animal species to another. The dog during the winter months follows very closely the pattern of man, but in the hot summer months the studies on dogs are of no value. We have not yet found a single substance or combination of substances sufficiently potent to eliminate the bacterial flora in the gastrointestinal tract of the dog in the hot summer months. This tremendous variation in activity with the seasons is not so evident in man although there does seem to be some increased resistance during the summer months to the elimination of the bacteria.

Intestinal Antiseptics. The antibacterial agents effective in the gastrointestinal tract can be divided into two groups, those which are bacteriostatic agents and those which are bactericidal agents. In the former group we have primarily the sulfa drugs and at the present time the effective members in this group are sulfasuxidine, sulfathalidine and phthalylsulfacetamide. We have studied approximately 50 different sulfa drugs to determine their effectiveness as antibacterial agents in the gastrointestinal tract. Of these 50, four have useful properties. For one reason or another only two, sulfasuxidine and sulfathalidine, have become practical intestinal antiseptics. There are a number of sulfa drugs which have some bacteriostatic activity in the gastrointestinal tract. Probably all the potent drugs of this group have some effect but it might be so small as not to be detectable or, as in the case of sulfathiazole and sulfa-

diazine, they have a selective activity as is illustrated by their prompt elimination of the shigella group of bacteria. There are certain organisms in the gastrointestinal tract which none of the sulfa drugs will inhibit, *i.e.*, the *alpha streptococcus fecalis*, which is always present in large numbers. This organism is ordinarily nonpathogenic. As will be seen later, there is a likelihood that the retention in the gastrointestinal tract of certain species of nonpathogenic bacteria is advantageous because, by their remaining in the gastrointestinal tract, the entire natural balance is not destroyed. Their natural antagonism prevents the outgrowth of undesirable higher plant forms.

The second group of antibacteria agents, those which are bactericidal in nature, include several antibiotics. Penicillin has no antibacterial activity but the author found it antagonized the action of sulfathalidine although it did not antagonize the antibacterial action of sulfasuxidine. Naturally, the bactericidal agents are of greater potential value than the bacteriostatic agents because by administration of the former group the organisms could be eliminated from the gastrointestinal tract much more rapidly than by the latter group. In the second instance, it would be necessary to eliminate all of the organisms present in the bowel as well as remove them from the surfaces of the bowel before they could be eliminated from the gastrointestinal tract, while in the first group contact with the antibacterial agent would bring about destruction of the viable organisms and not be dependent upon the mechanical removal or natural death of the viable organisms residing in the bowel when treatment was begun.

One of the first antibiotics studied was streptomycin. It was found to be rapid in its action to eliminate the *alpha Str. fecalis* as well as other bacteria in the gastrointestinal tract, but unfortunately resistant forms grew out very rapidly. Within 48 to 72 hours highly resistant organisms had reestablished themselves in the gastrointestinal tract. Dihydrostreptomycin follows essentially the same pattern. Bacitracin and chloromycetin are of little value. Aureomycin is found to be quite effective in eliminating most bacteria from the gastrointestinal tract. The undesirable property of aureomycin is that it causes nausea and vomiting too frequently in patients who are being prepared for gastrointestinal surgery. Also Monilia infections have been known to follow the administration of aureomycin. Terramycin has also been found to be fairly effective as an antibacterial agent but our experience with this drug has been highly unsatisfactory because it causes bloody diarrhea and the development of ulcers in the gastrointestinal tract probably due to yeast infections.

The most effective antibiotic we have encountered is neomycin. Neomycin is not absorbed from the gastrointestinal tract. Neomycin when given parenterally is fairly toxic, and this toxicity is such that it would preclude its use except under rare conditions. When given by mouth, however, the amount absorbed is so low that we have not encountered a single substance of toxic reaction even though this antibiotic has been administered to some 350 different individuals for periods as long as three months. In most instances, neomycin would completely eliminate bacteria from the gastro-intestinal tract within 24 hours. Following the elimination of bacteria, yeast begins to grow out in large numbers. In our rather extensive experience we have not encountered a single instance of yeast infection nor have we encountered an instance of a bloody diarrhea or ulcers in the gastrointestinal tract developing following the administration of neomycin. We have removed a large number of operative specimens which have been examined pathologically and there has been no evidence of secondary ulceration. In our experience, neomycin fails to inhibit the growth of Aerobacter aerogenes in approximately 10 per cent of the patients to whom the antibiotic is administered. This observation caused us to combine neomycin with sulfathalidine.

Combination of Antibacterial Agents. Following our initial observation that penicillin antagonizes the action of sulfathalidine, we have studied a number of combinations and have always been on the lookout for possible antagonism of the various agents. Neomycin apparently can be given with the sulfa drugs without antagonism. There does not appear to be any particular potentiation of either individual substance in the administration of this combination but there does seem to be a simple additive action when using the two compounds together. It has been observed rather frequently that when one substance is given and a resistant type of organism develops in the gastrointestinal tract, that organism would usually be resistant to other agents. For example, we found that streptomycin permitted rapid development of highly resistant forms of organisms. The administration then of sulfa drugs failed to lower the bacteria count. If the sulfa was given initially along with the streptomycin, the streptomycin did not counteract the action of the sulfa compound in any manner.

One of the most striking antagonisms we have encountered is between the two antibiotics, bacitracin and neomycin. Bacitracin has the property of almost completely inhibiting the bactericidal and intestinal antiseptic action of neomycin when these two substances are administered to dogs.

Healing of Bowel in the Presence of Intestinal Antiseptics. Whenever intestinal antiseptics are administered, it must be determined that they will not interfere with the normal healing of the gastrointestinal tissues and certainly they should not give rise to development of ulcers as has been observed in the case of terramycin. It is imperative that each and every agent which is to be used as an intestinal antiseptic must be studied experimentally to determine whether or not it will interfere with tissue repair. A single tragedy resulting from the use of a substance interfering with the tissue healing could undo many years of useful endeavor. We have demonstrated that sulfasuxidine and sulfathaladine, streptomycin and neomycin do not interfere with the normal processes of healing of the bowel tissues when given singly or in combinations.

The Problem of the Yeast. With the introduction of the bactericidal antibiotics such as neomycin, aureomycin and terramycin, it has been found that when practically all bacteria are eliminated from the gastrointestinal tract, the yeasts grow out uninhibited and in great numbers. On occasions they give rise to undesirable complications. Monilia infections have been observed following the administration of aureomycin. In this hospital we have observed the development of a bloody diarrhea with ulcers in the gastrointestinal tract following a brief period of administration of terramycin.

Although neomycin lowers the bacterial flora to a greater degree than any of the other antibiotics I have studied, and although the yeasts grow out in large numbers when the bacterial flora is eliminated, we have had no complications develop. This observation we cannot explain if the complications are due to the presence of yeast only. Probably other factors are involved. The question arises as to whether or not it is desirable to remove the nonpathogenic organisms from the gastrointestinal tract. So long as bacteria are present, the yeasts are held in check. The implantation of a resistant strain of nonpathogenic bacteria into the gastrointestinal tract might well be a desirable procedure.

Effect of Intestinal Antiseptics on the Clotting Mechanism. It might be anticipated that profound retardation of growth of bacteria in the gastrointestinal tract would interfere with local synthesis of certain vitamins including vitamin K with subsequent prothrombin deficiency. There have been published reports of such findings. We have been unable to confirm these findings. Following the prolonged administration of sulfasuxidine, sulfathalidine or neomycin, or combination of these drugs, we have been unable to demonstrate any alteration of clotting, bleeding or prothrombin

times when well controlled studies are done. We have not encountered increased bleeding following the administration of the above intestinal antiseptics to a large number of patients during the past 10 years whenever the patient is taking food. There is a possibility of alteration of the clotting mechanism if the patient is not taking food or is receiving a purified diet such as a protein hydrolysate.

Vitamin K is not given with these intestinal antiseptics unless the patient is found to have a prolonged prothrombin time or increased bleeding tendency, has been taking an inadequate diet or is receiving a purified diet.

Rapid Sterilization of the Gastrointestinal Tract. In instances of urgent surgery, we cannot prepare the gastrointestinal tract before operation. We have demonstrated repeatedly that if the gastrointestinal tract is filled with 1 per cent neomycin solution, as much as 1,000 cc. being instilled into the entire gastrointestinal tract, within 45 minutes no organisms can be cultured. We have operated upon these patients and so far have encountered no untoward effects. Caution should be directed to the fact that not more than 100 cc. of the 1 per cent solution of neomycin should be spilled into the peritoneal cavity because by this route neomycin in larger doses might prove to be toxic. We have repeatedly demonstrated the effectiveness of neomycin in peritoneal soiling. A 20 Gm. mouse has been injected with 1 cc. of a 10 per cent suspension of ordinary human feces. The control animals so injected will die within 24 hours. If within 30 minutes an adequate, tolerated dose of neomycin is administered intraperitoneally, these animals will survive. This experiment corresponds to the introduction of 400 Gm. of feces into the abdominal cavity of an 80 Kg. man for which a tolerated dose of neomycin could be given to protect the individual from this tremendous amount of contamination. The experiment was also conducted where the feces of the human which had been altered by the administration of neomycin was injected into the peritoneal cavity of the mouse in the same dosage and these animals survived without showing any untoward effects.

DISCUSSION

It has been amply demonstrated in numerous clinics and by individuals that intestinal antiseptics are effective in lowering or practically eliminating the occurrence of peritonitis following gastrointestinal surgery. In the past 10 years we have had neither leakage from a suture line nor the development of peritonitis in any patient postoperatively. No fecal fistulas have developed. All gastrointestinal surgery is now performed by the open method. It is no longer

necessary to resort to the more inaccurate so-called aseptic operative procedures of intestinal suture. An open anastomosis permits us to make a more accurate suture and better preserve the all-important blood supply of the involved tissues.

The most effective single agent in my experience with intestinal antiseptics is neomycin. The combination of neomycin with sulfathalidine has made an almost ideal intestinal antiseptic. The toxicity of this combination is that of sulfathalidine which is extremely low.

The complication recently introduced by the overgrowth of yeasts must be overcome. Either a satisfactory agent must be found to control the growth of these fungi or nonpathogenic bacteria which will suppress this secondary growth must be maintained in the bowel. This condition is met by the sulfa drugs which suppress the pathogenic bacteria but allow the nonpathogenic *alpha Str. fecalis* to remain and inhibit the overgrowth of the yeasts.

The use of neomycin to modify the intestinal flora when surgery is urgent should serve a real need.

THE EMBRYOLOGY OF THE MIDGUT AND ITS
RELATIONSHIP TO MIDGUT ANOMALIES

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T HE purpose of this paper is to review the development of the midgut. There are many midgut anomalies, though individual anomalies are infrequently encountered during the general surgeon's lifetime; consequently, the recognition of the anatomy of any encountered midgut anomaly depends primarily upon an active awareness of the normal embryologic process and secondarily upon an awareness of the varied anatomic results of abnormal or arrested midgut development. Such active awareness of seldom encountered pathologic entities is difficult to maintain during the long intervals between encountered cases. Further, anomalies of the midgut often are presented as acute surgical emergencies demanding immediate recognition without recourse to leisurely review. Upon these facts rests the sole justification for this paper.

The blood supply of the abdominal gastrointestinal tract is derived chiefly from the celiac axis, the superior mesenteric artery and the inferior mesenteric artery. We are concerned in this paper with that part of the gastrointestinal tract supplied by the superior mesenteric artery and its branches, and arbitrarily designate this region as the midgut. Thus the midgut in the developed state extends from the proximal second portion of the duodenum, supplied by the first branch of the superior mesenteric artery (inferior pancreaticoduodenal artery) to the splenic flexure of the colon, supplied in part by the midcolic branch of the superior mesenteric artery. Around the superior mesenteric artery as an axis, the rotational changes of the midgut occur. The artery presents a relatively fixed point from which to orient developmental change of the midgut region.

EMBRYOLOGY

The description of developmental processes often necessitates the use of artificial and not entirely accurate aids. For example, the staging or phasing of midgut development is an artificial way of describing a continuing developmental process. Midgut "rotation" actually may be a misnomer, inasmuch as the final intestinal position is a result of embryo growth as a whole, not basically of isolated

twisting of intestinal loops. I shall attempt to present the complicated midgut developmental process as a continuing one, with full realization of the inaccuracies involved in describing a picture, particularly a developing picture, in words.
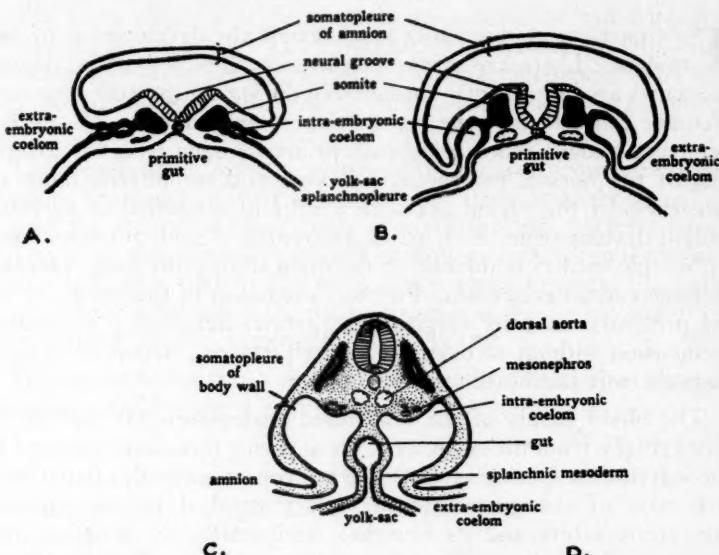

Fig. 1. Development of celoms and mesenteries and separation of primitive gut from yolk sac. Reproduced from Bradley and Patten, "Human Embryology," page 500, fig. 299.

Soon after the formation of the original blastocyst,[8] the three primitive germ layers of ectoderm, mesoderm and entoderm, form (fig. 1a). By a process of infolding of both intraembryonic and extraembryonic portions of somatopleure (ectoderm and somatic mesoderm) and splanchnopleure (entoderm and splanchnic mesoderm), the confluent primitive gut and yolk sac are separated from each other (figs. 1b, 1c). In this process dorsal and ventral mesenteries and intraembryonic coelom are formed. The ventral mesentery largely disappears, producing a unilocular celom. The dorsal mesentery persists as the intestinal mesentery. In figure 1c is shown the remainder of still unobliterated ventral mesentery and the connected gut and yolk sac in the region of the umbilicus. This connection is the omphalomesenteric (Vitelline) duct and normally is obliterated at the fifth week. The midgut viewed longitudinally at this point is a straight tube (fig. 2a).

Development of the intrinsic intestinal layers and structures be-

gins early. A stage of epithelial proliferation with plugging and subsequent vacuolization and lumen reformation occurs.

The further development of the midgut is illustrated in figure 2. All drawings are in left longitudinal oblique view. The process is continuing and progressive. The word rotation will be used to denote changes of the midgut around the superior mesenteric artery
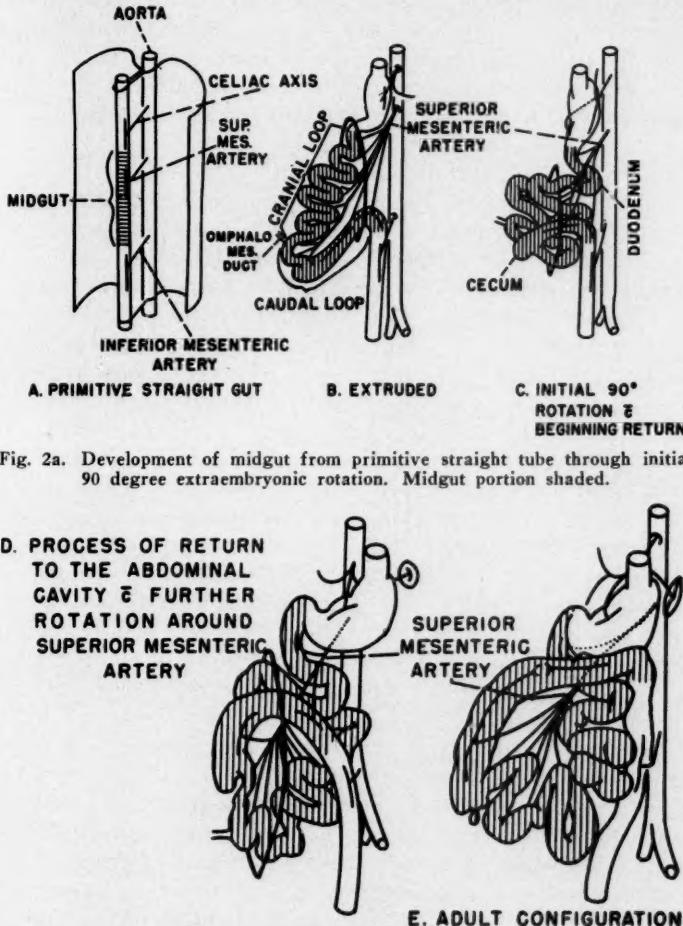
Fig. 2a. Development of midgut from primitive straight tube through initial 90 degree extraembryonic rotation. Midgut portion shaded.
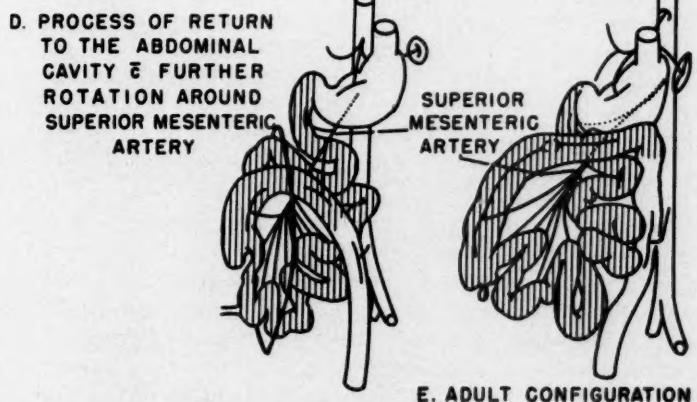
Fig. 2b. Continuing development of midgut with further rotation and return into the abdominal cavity. Midgut portion shaded.

axis, realizing that rotation may be a misnomer as regards the primary relation of the midgut to the developing embryo.

In figure 2a are shown the primitive straight intestinal tube and

the blood supply of the midgut from the superior mesenteric artery and its branches. This primitive tube grows rapidly and becomes coiled. Further, it grows more rapidly than the primitive abdominal cavity and therefore is extruded into the extraembryonic celom in the region of the umbilicus (fig. 2b). Differentiation into the future large and small bowel segments is also begun here. The mesenteries, not shown in figure 2, develop and differentiate with the bowel segments and, of course, contain their vascular supply. At the point shown in figure 2b the midgut is a relatively simple midline loop separated into a superior cranial limb and an inferior caudal limb by the fibrous remnant of the omphalomesenteric duct. The cranial loop is destined to form most of the small bowel and the caudal loop, the distal ileum, cecum, ascending and transverse colon. Continued growth results in beginning of rotation, which is associated with reassumption of intra-abdominal midgut position. This process is a continuing one and takes place in a counterclockwise manner around the superior mesenteric artery, thus placing the most proximal portion of the midgut posterior to the superior mesenteric artery (fig. 2c). The cranial loop is placed toward the right and the caudal toward the left. The enlarging abdominal cavity begins to encompass or makes possible retraction of the midgut loop, as the case may be, at this point. Probably due to shortness of the proximal inferior pancreaticoduodenal branch of the mesenteric artery, the most proximal part of the cranial loop enters the abdominal cavity first. More distal portions successively follow by a process of continuing counterclockwise rotation. If the surgeon will imagine that he is replacing the bowel, segment by segment, beginning at the proximal end of the midgut loop as seen in figure 2c, into the abdominal cavity, it will be clear that such replacement logically produces a further counterclockwise rotation about the superior mesenteric artery. The large bowel forming loop will be replaced last and will therefore be in an anterior position in relation to the superior mesenteric artery. The position will thus be approximately that seen in figure 2(continued)d. The embryo's growth progresses primarily in a cranial direction. This growth, along with further development and segmentation of the cecum, ascending and transverse colon, results in the adult large and small bowel positions seen in figure 2(cont.)e and gives us a continuing picture of further rotation around the superior mesenteric axis.

We have thus in summary a continuous counterclockwise rotational process of somewhat over 360 degrees resulting in a retroarterial duodenal position and a prearterial inverted U large bowel loop. The left side of the U consists of the proximal hindgut segment pushed over by developing small bowel. The midgut is in the

extraembryonic position from about the fifth to the tenth week. The final attainment of adult bowel position may be reached at or even after embryonic maturity. Study of the excellent description and illustrations of midgut development found in Callander's first edition[1] should prove to be of marked help to the surgeon in further orienting himself.

Fixation. Normally, according to Callander,[1] after completion of rotation the cecum, ascending colon and ascending mesocolon fuse with the right posterolateral parietal peritoneum, thus fixing these structures and the underlying duodenum at the hepatic flexure. Excessive cecal fixation makes exposure of the appendix more difficult and variations in cecal fixation may result in folds forming fossae into which bowel may herniate.

At the termination of rotation the duodenojejunal junction is placed against the transverse mesocolon and fuses there. Variations in fusion in this region again may produce fossae which may be the site of intestinal herniation.

Anomalies resulting from abnormal midgut development. Midgut anomalies are conveniently separated into: (1) anomalies related to intrinsic development; (2) anomalies related to omphalomesenteric duct development and (3) anomalies related to midgut rotation and fixation. An attempt will be made to briefly point out those anomalies more commonly requiring surgical treatment, and to relate them to the developmental process.

Anomalies of intrinsic development. The principal anomalies of intrinsic development are intestinal atresia[3] and intestinal duplication.[4] Evans[3] presents evidence that intestinal atresia may be related to extrinsic rather than intrinsic developmental pathology. Conversely, defects concerned with arrested rotation may primarily be due to intrinsic defect of the midgut's inherent fixation potential, rather than to defect in the process of rotation as commonly accepted.

Anomalies related to omphalomesenteric duct development. These anomalies are well presented by Cullen.[2] The omphalomesenteric duct may persist as a patent tract to the umbilicus, giving rise to an umbilical intestinal fistula with or without prolapse of contiguous ileum through it. It may persist as a fibrous cord with completely obliterated lumen and act as an inciting cause of intestinal obstruction, or its lumen may persist in part, giving rise to umbilical cyst or sinus. Persistence of the proximal portion results in Meckel's diverticulum.

Anomalies related to midgut rotation and fixation. Numerous

anomalies occur from defective midgut rotation and fixation. I shall merely attempt to relate some of them to this progressive process and point out those producing dysfunction and which are thus of surgical importance.

Failure of the midgut loop to become reincorporated in the abdomen at the tenth week results in omphalocele, a condition demanding immediate surgical intervention. Omphalocele may contain any or all of the midgut loop including attached liver and pancreas. It may be necessary to cover large omphaloceles with skin only, to prevent the complications of increased intra-abdominal pressure occasioned by layer closure.[6] In a recently observed case the midgut was in a position of completed normal rotation showing that rotation may proceed outside the abdominal cavity.

If the midgut reenters the abdominal cavity without rotating further than the initial counterclockwise twist, the cecum, ascending and transverse colon will all be found on the left. The small bowel will be on the right, and the duodenum will be parallel rather than posterior to the superior mesenteric artery. Such a position is asymptomatic and has its anatomic normal counterpart in the intestinal arrangement of the cat. Further, it is the position to be produced surgically in treating duodenal obstruction from compression of the duodenum by a right upper quadrant cecum.[7]

In the continuing rotational process, arrest and fixation may occur when the cecum has reached the right upper quadrant position. Extrinsic duodenal obstruction may result from compression of the fixed cecum or a cecal fixation band upon the duodenum. Arrest in this position may also be associated with volvulus of the small bowel because of the narrow area afforded for fixation of small bowel mesentery.

After completion of rotation, the right lower quadrant cecum may fail to become fixed and as a result may twist as a volvulus. Folds forming fossae which produce sites for internal hernia may also result from the fixation process. These fossae occur most commonly at the cecum and at the duodenojejunal junction. Various anomalies, such as complete herniation of the small bowel into its own mesentery, may result from abnormal rotation. Complete reversed rotation results in transposition of the colon and duodenum in relation to the superior mesenteric artery. A number of more common rotational and fixational anomalies are well illustrated and discussed by Gardner.[8]

SUMMARY AND CONCLUSIONS

1. The normal development of the midgut has been followed from the point of differentiation of germ layers through the forma-

tion of a straight intestinal tube and thence through its rotational and fixational processes to the normal adult anatomic state. This process, which occurs in both the intraembryonic and extraembryonic celoms, has been treated as a continuing one. The importance of the superior mesenteric artery and its branches as an axis of rotation has been emphasized, and the midgut has been defined as that portion of the primitive intestinal tube supplied by the superior mesenteric artery and its branches.

2. It is believed that the surgeon can best maintain an active knowledge of the developmental anatomy of the midgut by considering the rotational process as a continuing counterclockwise spiral rotation through approximately 360 degrees. Such active knowledge is essential to the recognition of the anatomy of congenital anomalies of the midgut, in as much as any single anomaly is infrequently encountered by the individual surgeon.

3. Many anomalies of arrested or abnormal midgut development occur. Some of those of primary surgical importance are briefly discussed in relation to their mode of development.

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STANDARDIZATION OF GASTRIC RESECTION IN THE TREATMENT OF PEPTIC ULCER

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W E take no issue with the premise that the management of peptic ulcer is primarily the concern of the internist, but we are very cognizant of the well established fact that about 10 per cent of those suffering from duodenal ulcer and a much higher per cent of those with the gastric variety of the disease must seek the aid of the surgeon if they are to control satisfactorily their disease. Acute perforation, penetration, obstruction, hemorrhage, failure of medical management, and the ever present danger of malignancy in gastric ulcer are the complications that demand surgical consideration. Through the past few decades simple gastrojejunostomy, pyloroplasty, and lately vagus interruption have had their rise and fall of popularity, and it is without doubt that all of these operations have controlled otherwise unmanageable ulcers. However, I do not believe that many will take issue with the stand that partial gastrectomy is the most popular and has the most to offer in the overall surgical treatment of peptic ulcer. Accepting this last statement, we will limit the rest of this presentation to a discussion of partial gastric resection.

Direct and indirect investigation of the gastrectomized ulcer patient reveals that there is much to be desired in the end results of some of these patients. On the other hand the majority seem to be entirely or almost entirely free of all gastric symptoms and are able to carry on lives of normal activity without the host of limitations that is the lot of the patient with severe ulcer. Review of the literature and discussions with surgeons reveal that there is a whole host of variations in the technic of gastrectomy for ulcer. Many of these variations are based on good physiological principles, experimental investigation, and satisfactory clinical results. Others are not. Why is it that some internists look on ulcer surgery as the last resort while others who see the good results are always ready to offer it to their complicated ulcer patients? Some surgeons must do a better job than others. What technical variations are responsible for high mortality, long periods of morbidity, the so-called "gastric cripples," and an appreciable incidence of postoperative ulcers? These variations should be better understood. In the past two decades an enormous amount of research has been carried out and presented in the literature concerning the physiology of peptic

ulcer. We believe that more surgeons should incorporate these experimental results in their application of gastrectomy and, after studying their clinical results, should present evidence to substantiate the variations in technical steps that they believe should be incorporated in a more standardized gastric resection for the treatment of peptic ulcer.

It is our purpose in this paper after evaluating the physiological and experimental evidence at hand and correlating it with the postoperative study of a series of personal cases, to present what we believe is the most satisfactory method of management of the technical steps in this operation.

Before proceeding further we wish to establish what we believe are the desired end results to be attained with the operation in question. Our operation must result in (1) a low mortality, (2) a low morbidity, (3) few postoperative gastric cripples, (4) a very low incidence of recurrent ulcer, and (5) a patient who can return to normal living without the burden of a strict ulcer regimen. A careful study must be made as to what constitutes the basis of these desired end results. First we must consider the causes of postoperative deaths as far as the technic is concerned. We are not concerned with the systemic causes of death, such as embolism, cardiac failure and pneumonia, in as much as they are not primarily related to the technic of the operation. For our consideration, then, peritonitis assumes the most importance. This is due to duodenal stump leakage, less often anastomotic failure, and occasionally to injury to the bile ducts in the removal of a particularly extensive ulcer. Hemorrhage, most often from a vessel in the anastomotic line, is the other important cause of postoperative death.

What are the causes of morbidity that can be related to technic? We must understand the causes of gastric retention and stomal obstruction. We must also consider factors which prevent an early return to normal living. We must have a postoperative stomach that will early tolerate adequate feeding.

Next we must consider the postoperative "gastric cripple." He has the persistent "dumping syndrome." He suffers from nausea and vomiting of bile. Weakness and anemia dog his footsteps and he cannot maintain his weight. What technical failures are responsible for these situations?

If our incidence of recurrent marginal ulcer is high, then we are not making the most of our opportunities to modify the physiology that affects the course of our ulcer diathesis. Our procedure must satisfactorily reduce the acid gastric secretions and provide an intestinal mucosa at the stoma that is most resistant to ulceration.

If we can satisfy the above criteria we will usually be rewarded with a patient who can successfully take his place as a productive economic unit in society.
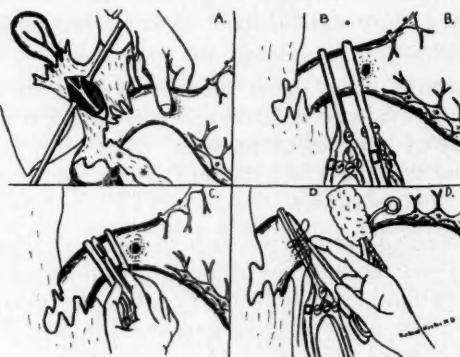

Fig. 1. Routine management of the duodenal stump. (A) The surgeon must be ever mindful of the course of the common bile duct. Its injury is a real danger in an active penetrating ulcer. (B, C) Division of the duodenum is carried out between clamps beyond the ulcer if that area is readily mobilized. (D) Then closure of the stump is begun over the clamp.
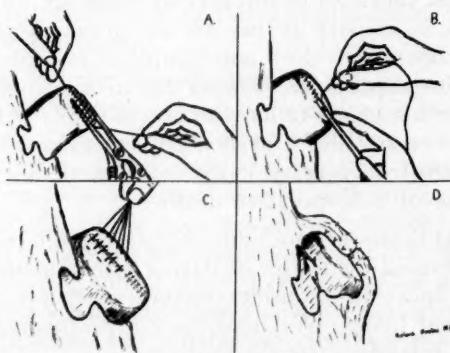
Fig. 2. Routine management of the duodenal stump (continued). (A) Removal of the clamp following the placing of the first layer of continuous chromic catgut through the seromuscular layer. (B) The second row is a continuation of the first and is also carried through the seromuscular layer. (C) The final row of closure is of interrupted mattress stitch with nonabsorbable suture. (D) Neighboring tissues are tacked over the closure as available.

From the above we can deduct that the following are the important steps in gastrectomy for peptic ulcers: (1) management of the duodenal stump, (2) extent and anatomy of resection, (3) management of the anastomosis, and (4) early postoperative regimen.

Leakage from the duodenal stump with subsequent peritonitis,

fistulas, and postoperative death is usually due to a combination of two factors, namely, increased intraluminal pressure behind the closure and inadequate blood supply to the suture line. We believe
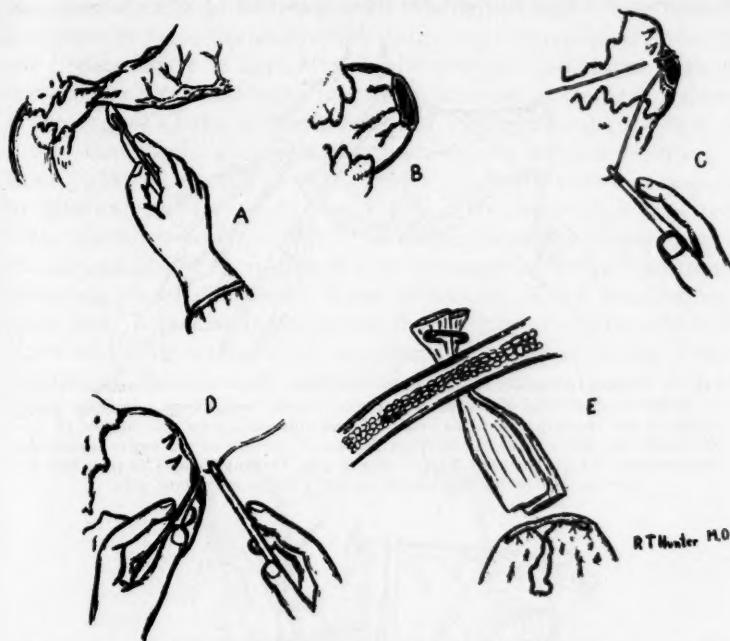
Fig. 3. Alternate management of the stump in severe sclerosing ulcer. (A) The duodenum is severed at its narrowest point which is usually through the ulcer. (B) The small opening in the duodenal stump is shown ready for closure. A minimum of mobilization is carried out, thus protecting the bile ducts and insuring a good blood supply. (C) The first row of interrupted sutures is placed through the seromuscular layer. (D) The closure is completed with a second layer which incorporates protective tissues as available. (E) Drainage of the area is always carried out when there is the least question of security of the closure.

the actual technic of closure is of secondary importance. Increased intraluminal pressure in our opinion is most often due to partial or complete obstruction of the afferent loop at the gastroenterostomy. Attention to the placing of the stoma and protection of the duodenal blood supply assume most importance. Removal of the ulcer itself seems desirable if it can be readily accomplished. This is of course particularly true if there is active hemorrhage from the lesion at the time of surgery. However, we are in complete accord with the opinion of Eastman and Cole[1] that it is not necessary to remove the ulcer. We do feel that it is hazardous to leave a sclerosing ulcer in place and thus set the stage for a closed loop obstruction between

the line of closure and the edematous postoperative ulcer. It is readily apparent that that situation can rapidly lead to leakage of the duodenal stump and its serious sequelae. Then what must be our course of action in the sclerosing, penetrating ulcer whose com-
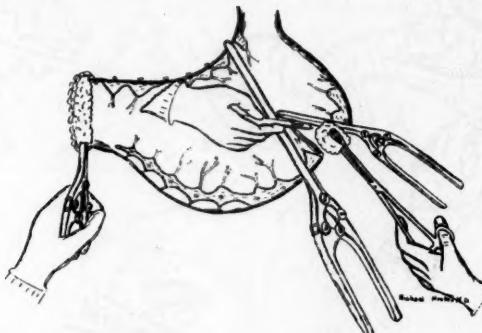
Fig. 4. Illustration shows the extent of resection. Every trace of antrum must be included. Inclusion of the duodenal ulcer is not mandatory. At least two-thirds of the stomach should be removed and this should include nearly all of the lesser curvature so as to interrupt the major portion of the vagal supply to the stomach. The segment of gastric stump used for anastomosis is included in the small Payr clamp placed on the greater curvature side.
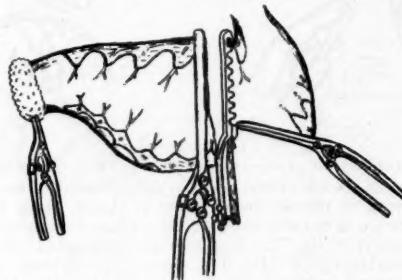
Fig. 5. The resection is completed. A Furniss clamp is shown in place over the portion of the gastric remnant that is to be closed.

plete removal would jeopadrize the bile ducts? In this situation, as suggested by Waugh,[2] we have on repeated occasions severed the duodenum through the ulcer at its narrowest point freeing up only sufficient tissues for the most rudimentary closure. Then the opening of our duodenum to be closed will be of the smallest caliber, the blood supply will be adequate, and the danger of damaging the extra hepatic biliary system will be negligible. It is well in these cases and in any other where there is the slightest question about the integrity of the stump closure to carry out postoperative drainage of that area.

There has been considerable discussion in the literature as to the extent of resection that is the most advisable. It is the opinion of most gastric surgeons that between two thirds and four fifths of the stomach should be removed. We believe the lower figure is adequate in the older individual with a burned out ulcer, whereas, the higher figure is necessary in the younger individual with the more active ulcer. As important as the amount of stomach removed is the region of the stomach removed. Edkins[3] first proposed the theory that fundic secretion was stimulated by a hormone which he named gastrin, which is produced by the antral mucosa in response to chemical stimulation by foodstuffs. This theory has repeatedly been substantiated by others.[4,5] Wangensteen[6] has commented on the advisability of sacrificing the major vagal nerves in resection by removing almost the entire lesser curvature of the stomach over which they began their course on that organ. To summarize this phase of the discussion then, we must remove approximately three fourths of the stomach sacrificing the lesser curvature and the entire pyloric antrum.
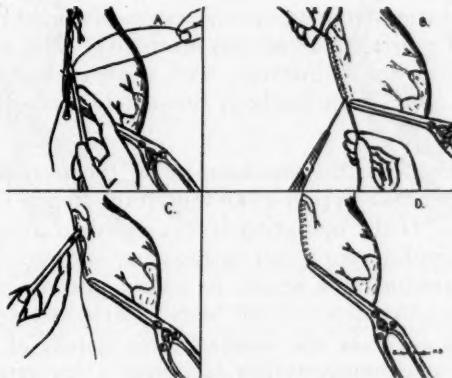
Fig. 6. (A, B, C, D). The successive steps in the 3 layer closure of the lesser curvature end of the gastric stump are illustrated.

In the discussion of the management of the gastrointestinal stoma, we are concerned chiefly with its size, its position in relation to the transverse colon, and the length of the afferent jejunal loop. We have gradually cut down the size of our stomas from the full width of the gastric stump to a size of about 4 to 5 cm. The rapid emptying of the stomach with its unmixed food which is certainly a major factor in the "dumping syndrome" is avoided with the smaller opening. In forming the small stoma it is necessary to close a portion of the gastric cuff after the method of Hofmeister. We

see no sound reason to make a gastroenteric stoma that is any larger in diameter than the loop that it feeds. With the smaller stoma the bile and duodenal secretion are more likely to stay in the jejunum rather than to pool in the stomach and cause gastric symptoms. Many excellent surgeons advocate that the anastomosis be placed anterior to the transverse colon but it is our feeling that an anastomosis carried through the mesocolon is more satisfactory. It has been repeatedly demonstrated[6,7] that the resistance of the intestinal mucosa to acid pepsin decreases progressively as the distance from the stomach increases. The work of others[8] suggests that the closer the anastomosis is to the stomach, the better the efficiency of digestion. This evidence and the fact that we believe that the posterior anastomosis functions more satisfactorily make it the method of choice in our resection. It seems unnecessary to mention the necessity of placing the anastomosis so that there is no possible kinking of the loops. Much has been written about the superiority of the closed anastomosis. To us it is a blind procedure and with its use the hemostasis at the suture line cannot be as certain. The evils of contamination are not substantiated. We have never seen a case where contamination from an open anastomosis could be implicated as the cause of mortality or serious morbidity. The necessity of a narrow cuff so as not to interfere with gastric emptying or cause proximal loop obstruction has been adequately expounded by Wangensteen.[9]

We have considered the management of the stomach in the immediate postoperative period as an important step in our standardized procedure. If the operation is accomplished properly, we see no reason to employ continuous intragastric suction. If the stoma is open and functioning it serves no useful purpose and can only deplete the chemical stores of the body. Early and adequate feeding started as early as the stomach will handle it seems most rational. The anastomosis is just as strong a day after surgery as it is a week later. A quick return to an adequate diet insures a shortened convalescence.

This presentation is based on a series of 50 consecutive personal cases of peptic ulcer in which gastric resection was carried out as the primary operative procedure directed at the ulcer. The series is too small to warrant statistical study but analysis of the results does give support for the method of gastric resection presented. The operation was carried out for duodenal ulcer in 36 instances and for gastric ulcer in the remaining 14. There was no operative or postoperative mortality. Seven of the patients were women and the rest were men. Two of the gastric ulcers showed areas of malignant change. The operation carried out was essentially that which

will be presented except for variations as indicated. In 3 of the cases of gastric ulcer the gastric stump was anastomosed to the open end of duodenum (Schumacher modification of Bilroth I) with good results. In 2 of the early cases of the series the entire gastric
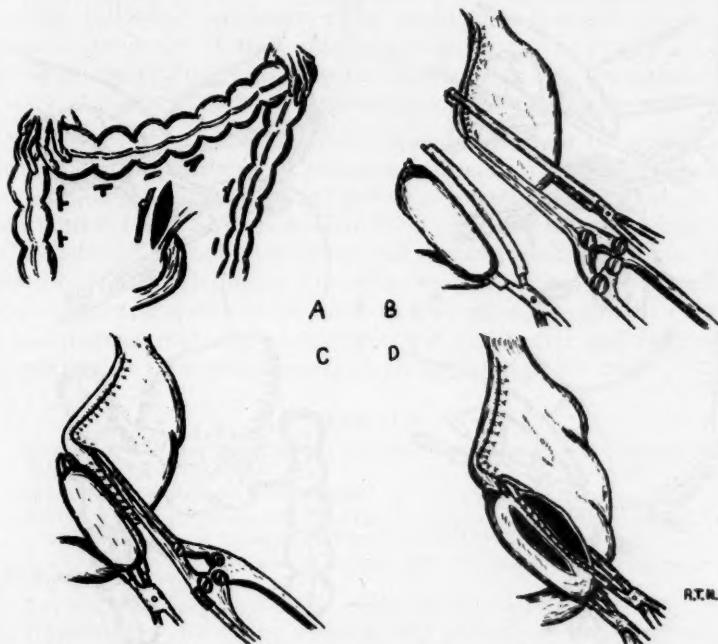
Fig. 7. (A) The opening in the transverse mesocolon for the anastomosis is illustrated. This must be placed so that a very short afferent jejunal limb will be available. Its position and direction must be such that there is no kinking or twisting at the anastomosis. (B, C, D) The first steps in the open anastomosis utilizing rubber shod clamps is illustrated. The first posterior row of non-absorbable suture is placed before the jejunum and stomach are opened.

stump was anastomosed to the jejunum after the method of Polya. In 1 case the removal of the antral mucosa was incomplete because of technical difficulties combined with the poor condition of the patient. Most of the patients were out of bed the first or second day following surgery. Forty-three of the series of 50 cases are either under observation, have responded to a recent questionnaire, or were followed for more than a year following surgery. As far as is known, none of these cases has been subjected to further surgery on the stomach. One case has had a diagnosis of jejunal ulcer made elsewhere. He was the case in which the entire antral mucosa was not removed. There were no postoperative duodenal fistulas

or known leakage of the duodenal stump although drainage of that area was carried out as a precautionary measure in several cases. There was no significant postoperative bleeding. No attempt has been made to keep these patients on any dietary regimen other
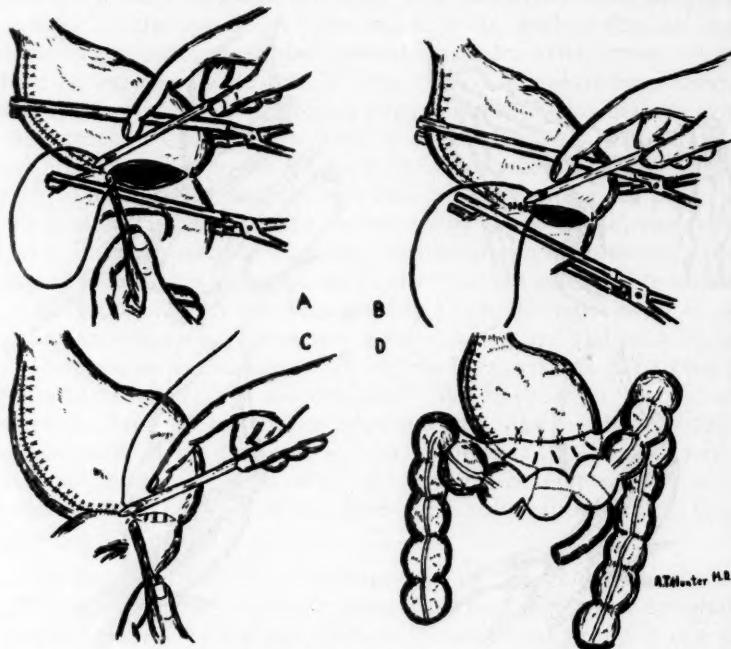
Fig. 8. The anastomosis is continued. (A, B) The inside row of suture is of chromic catgut carried through all layers and it serves as a hemostatic stitch. (C) The anastomosis is completed anteriorly with interrupted nonabsorbable mattress stitches. (D) The stoma is placed below the opening in the mesocolon. The latter is stitched to the stomach.

than to avoid foods that upset them and to take extra feedings particularly during the first few postoperative months. Follow-up reveals that 39 of the 43 traced cases have no significant dietary or gastric complaint, follow no particular regimen, and carry out activities commensurate with their age and general condition. Two cases have moderate postprandial symptoms that are partially incapacitating. The results in 2 others have been classed as unsatisfactory. One is the mentioned case of recurrent ulcer and the other is a persistent "dumping syndrome." Interestingly, he was one of the cases in which the entire width of the gastric stump was used for the stoma. Dumping syndrome has been a prominent but not too consequential postoperative symptom. However, it has disap-

peared in from a few weeks to a few months in all but three cases. It was noted that in the later cases in this series where the tendency has been to decrease the size of the anastomotic stoma that the "dumping syndrome" has been much less a problem. Most of the patients in this series have been unable to maintain their normal weight following operation. The usual loss averages about 10 pounds and is not of much significance, so far as the patient's well-being is concerned. This is most likely due to the decreased efficiency of digestion in the postgastrectomized individual.

Figures 1 through 8 and their legends illustrate the important steps in a method of gastric resection for peptic ulcer which is based on physiological principles and personal experiences, and which to this author could serve as a basis for a standardized procedure. It is hoped that in the future there will be more contributions to the literature substantiating or disproving the variations in the technical steps of gastric resection for ulcer so that we may gradually evolve a sound standardized procedure that will give better and more permanent relief to the sufferers from complicated peptic ulcers.

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TRAUMATIC PANCREATITIS*

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THE pancreas, in spite of its deep and protected location, is sufficiently subject to trauma so that injuries to it and their sequelae are surgically significant. Traumatic involvement of the pancreas is not readily apparent, and its recognition is difficult. It does occur, however, with sufficient frequency to be considered in a patient with abdominal injury. Injury to the gland may or may not be associated with injury to important adjacent viscera. It is imperative that the surgeon be aware of pancreatic injury before undertaking surgery upon the traumatic abdomen. With the better results being obtained by the conservative management of pancreatitis, regardless of type, the preoperative diagnosis of this condition becomes even more important.

The mechanism for injury to the intra-abdominal organs surrounds us in our everyday life, especially with the high incidence of automobile and industrial accidents. Injury may be produced by direct or indirect violence, by crushing or penetrating wounds. Pancreatic injuries most often are the result of sudden blunt violence to the upper abdomen, this trauma forcibly wedging the organ against the bodies of the vertebrae. The tissue of the gland is soft and easily bruised. Pathologic changes may vary from simple transient edema to immediate or late hemorrhage into the glandular substance. The hemorrhage may or may not be confined beneath the capsule of the gland.

Symptoms, of course, vary with the associated injuries. Penetrating abdominal wounds, massive and evident intra-abdominal hemorrhage, and ruptured hollow organs, as evidenced by pneumoperitoneum, require emergency, lifesaving surgery. Isolated pancreatic injuries do not produce such dramatic symptoms. Symptoms may not appear for hours or days following injury. The classic shock and prostration of acute pancreatitis are not characteristic, though abdominal pain is a constant finding. In 4 of the 5 cases observed within the past five years at the University Hospital it was the presenting complaint. In the other case abdominal pain was secondary to pain produced by associated fractures. In each case the pain was upper abdominal. In 3 of the 5 cases it was mid-epigastric but without characteristic bandlike radiation or radiation to the back. Nausea and vomiting were present in all cases. Shock

or signs of blood loss were not prominent features of the symptomatology in the uncomplicated cases.

Diagnosis in each case was established or confirmed by serum amylase determinations, with the interval between injury and diagnosis varying from six hours to six weeks. The serum amylase test has become a valuable diagnostic aid in acute conditions of the pancreas and serves well to indicate involvement of this organ following abdominal injury. By the time the patient is seen with acute abdominal symptoms due to pancreatic involvement, the serum amylase is almost always elevated to diagnostic levels. Exudation of amylase rich fluid into the peritoneal cavity is known to occur soon after any acute involvement of the pancreas. With this in mind, Keith[4] and Wright[16] have advocated peritoneal taps and the analysis of the obtained fluid for amylase in the diagnosis of acute conditions of the pancreas. In traumatic conditions of the abdomen especially, it may help to rule in or out intraperitoneal hemorrhage or ruptured hollow organs by disclosing blood or bile tinged fluid. The routine use of the serum or peritoneal amylase determinations following abdominal injury may establish a diagnosis in an otherwise obscure condition.

Few surgeons prior to the development of the amylase test refrained from exploring the abdomen in the presence of early symptoms called forth by this condition. Treatment in our opinion, however, should be conservative as in other forms of pancreatitis. Complications such as subsequent pseudocyst formation may require drainage.

The following cases of traumatic pancreatitis have been seen at the University Hospital within the past six years.

CASE 1. H. J. G., a 29 year old white male, was first seen in the emergency room of the University Hospital on Dec. 2, 1945, following a fight. He was complaining of a scalp wound and mild abdominal pain. The laceration was sutured. The patient refused to be admitted to the hospital for observation, although on examination he had moderate midepigastric tenderness and a heel mark in the midepigastrium. The following morning, however, he again returned, complaining of severe abdominal pain, nausea, and vomiting. On admission there was marked tenderness and guarding in both the upper quadrants and severe midepigastric tenderness below the zyphoid. Abdominal roentgenograms disclosed no free air under the diaphragm. On conservative therapy, symptoms rapidly subsided. No serum amylase studies were done on this admission and he was discharged ambulatory on his seventh hospital day.

Five days later he was suddenly seized with severe midepigastric pain and readmitted to the hospital. Signs and symptoms were similar but more intense than those found on his first admission. The serum amylase was elevated to 611 Somogyi units. An emergency exploratory laparotomy was carried out with the preoperative diagnosis of traumatic pancreatitis. At that time the body of the pancreas was found to be thickened and markedly edematous.

There was an increased amount of blood tinged serous peritoneal fluid and definite scattered areas of fat necrosis. The lesser sac was not remarkable; however, the retroperitoneal space contained old hemorrhage and fat necrosis. The area was drained.

Convalescence was uneventful. He was again seen in May 1946 with a two months' history of aching upper abdominal pain, backache and an enlarging abdominal mass. On examination there was a firm 12 cm. midepigastric mass which proved to be a pancreatic pseudocyst. The cyst was marsupialized and continued to drain for seven months. When last seen in September 1947, 21 months following injury, the patient had no symptoms referable to the gastrointestinal tract. The abdominal wound was well healed, and no masses were palpable.

In spite of the fact that drainage of the lesser sac was done, the patient subsequently developed a pancreatic pseudocyst. Whether symptoms would have progressed under conservative management is not known.

CASE 2. M. R. H., a 47 year old white male, was admitted to the hospital on Aug. 4, 1948, following an automobile accident. As the driver, he was thrown against the steering wheel of the car and suffered fractures of the surgical neck of the left humerus and the ninth and tenth ribs posteriorly on the right.

He was complaining of midepigastric pain. The patient was observed for 24 hours during which time a progressive increase in abdominal rigidity and distension was noted. There was no evidence of shock or massive blood loss. Upright roentgenograms of the abdomen on three occasions were negative for free air under the diaphragm. Progression of abdominal symptoms in spite of supportive and conservative measures forced an exploratory operation with the preoperative diagnosis of ruptured liver. No serum amylase studies were done prior to surgery. At surgery about 750 cc. of free peritoneal blood was found. There was a laceration of the transverse mesocolon and scattered areas of fat necrosis. The pancreas was completely transected as it passed over the lumbar vertebrae. From the tail of the pancreas appeared a structure which apparently was a completely lacerated pancreatic duct. No similar structure was found in the head segment of the pancreas. The duct was ligated and the pancreas repaired. The area was drained. During surgery the patient had what was thought to be a transfusion reaction. Immediately postoperatively the serum amylase was 1472 Somogyi units. The patient became oliguric, The B.U.N. (blood urea nitrogen) rose progressively, and he died on the sixth postoperative day. Autopsy permission was not obtained.

CASE 3. On Aug. 17, 1949, C. D., a 47 year old colored male, was admitted to the hospital with complaints of abdominal pain and swelling. Six weeks prior to admission, while changing a light bulb, he fell from a chair, striking the upper part of his abdomen on a banister. Dating from this event, he noted increasingly severe generalized abdominal pain. There was marked anorexia and occasional bouts of nausea and vomiting. He admitted losing 25 to 30 pounds in weight. He also noted progressive abdominal swelling. His previous health had always been good. On examination he appeared poorly nourished and chronically ill. The upper abdomen was distended by a tense fluid mass. The admission serum amylase was elevated to 1800 Somogyi units. With the preoperative diagnosis of traumatic pseudocyst of the pancreas, exploratory operation was performed. Upon exploration the abdomen was found to con-

tain about 2500 cc. of milky fluid. The Foramen of Winslow was closed by dense adhesions. The lesser sac was found to be distended by a pinkish fluid. The cyst was evacuated and marsupialized. Postoperatively the drainage was copious but gradually subsided so that only a small fistulous opening was present one month after surgery. This opening spontaneously closed within the month. On one year follow-up examination, the patient had gained 12 pounds in weight and had no digestive complaints. The wound was well healed and there were no abdominal masses.

CASE 4. H. D., a 32 year old white male, was struck in the midepigastrium by the handle of a file that kicked back from a lathe on April 3, 1950. He experienced dull, aching, midepigastric pain for about 20 minutes. This subsided only to recur five hours later. This later episode gradually increased in severity and was accompanied by nausea and vomiting. With these complaints, he was admitted to the hospital about 12 hours following his injury. On examination, the patient had localized and had rebound midepigastric tenderness with guarding. At no time was there evidence of shock or blood loss. Abdominal roentgenograms did not disclose free air. The serum amylase, which on admission was 378 Somogyi units, rose to a maximum level of 747 units on the third hospital day as noted in the figure. The urine amylase was greatly elevated (1900 Somogyi units). A barium swallow on the first hospital day showed an enlargement of the duodenal C-loop with compression of the second portion of the duodenum. This was interpreted as being due to pancreatic swelling. Subsequent gastrointestinal series demonstrated a gradual enlargement of the duodenal C-loop to three to four times normal size.
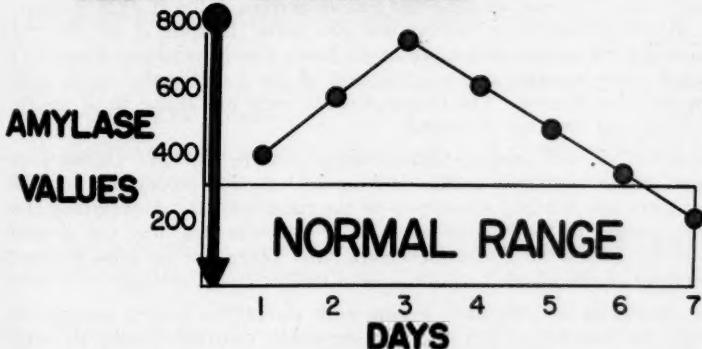
Fig. 1. H. D. Case 4. Note delayed rise of amylase from questionable levels to diagnostic levels.

The patient was initially treated by conservative means with gastric suction and daily bilateral splanchnic blocks for pain relief. On the sixth hospital day the patient developed clinical jaundice at which time a firm midepigastric mass became palpable. Gastric suction was maintained for one week during which

time the patient was acutely ill. Symptoms gradually subsided and the patient began to tolerate a diet. The epigastric mass rapidly decreased in size, and the jaundice disappeared. The mass was still slightly palpable at discharge, but the patient was asymptomatic after 23 hospital days. On a recent check, he was working daily and feeling well. The midepigastric mass was no longer palpable.

In this case the serum amylase was elevated to diagnostic levels within 16 hours following injury. An additional confirmatory test the urine amylase became even more significantly elevated.

CASE 5. C. S., an 18 year old Army private, was involved in an automobile accident on May 6, 1951, in which he was thrown from his car and run over by another automobile. On admission to the emergency room he was somewhat stuporous but complained of shortness of breath and severe abdominal pain. On examination he was cyanotic and in shock with a rapid, irregular pulse. There was a marked mediastinal shift to the right and signs of a left tension pneumothorax. Linear contusion marks extended across the upper abdomen and left chest. The abdomen was rigid and diffusely tender, especially in the midepigastrium and right upper quadrant. Shock was combated with plasma, periston (a plasma extendor), and whole blood. Following a closed thoracotumy with removal of blood and air from the left chest the patient's condition stabilized. He continued to complain of abdominal pain and vomited brownish bloody material. Chest roentgenogram showed fractures of the left seventh, eighth and ninth ribs in addition to the hemopneumothorax. Abdominal films showed no free air but a rather prominent gastric dilatation. Levine tube suction was initiated. A catheter was placed into the bladder and clear urine obtained. Because of the persistent abdominal signs and the large amount of blood necessary to maintain his blood pressure, it was thought that he was actively bleeding into his peritoneal cavity. A needle puncture of the peritoneal cavity was done about six hours after admission. Grossly bloody fluid was obtained that had an amylase value of 2128 Somogyi units. A concomitant blood amylase was 680 units. In spite of the elevated amylase value the patient was explored 12 hours after admission. The spleen was found to be lacerated and was removed. A small perforation in the gallbladder was also sutured. The retroperitoneal space was found to be grossly hemorrhagic and was not disturbed.

Postoperatively the patient became oliguric. The B.U.N. (blood urea nitrogen) rose precipitously and he died on the seventh postoperative day. At autopsy there was bilateral thrombosis of the renal arteries. Antemortem clot was also present in the adrenal veins. The pancreas was firm and showed extensive fat necrosis in the peripancreatic fat. There was no gross evidence of laceration of the gland.

The association of pancreatic lesions with thrombosis is well recognized. Although the outcome of this case was inevitable, constant bladder drainage may offer a prognostic sign in similar cases. Accurate knowledge of the urinary output is essential in regulating shock and fluid balance.

COMMENTS

The above cases well illustrate the type of trauma that may produce traumatic pancreatitis. Insignificant or remote trauma must not be taken lightly. A careful evaluation of the mechanism of in-

jury may point toward pancreatic involvement. The delayed onset of symptoms and the high incidence of pseudocyst formation in this condition have been noted in cases reported by other authors.[11,14] The striking similarity between the above cases and cases previously reported suggest a group of symptoms that should arouse a suspicion of traumatic pancreatitis. Symptoms initiated by blunt trauma to the upper abdomen and characterized by delayed onset, progressive signs of irritation in the upper abdomen, and lacking the signs of blood loss or pneumoperitoneum are extremely suggestive. Diagnosis can usually be established by serum amylase determination, thus allowing the patient at least a trial on conservative management. In questionable cases, peritoneal taps should be done without hesitancy. More cases of this condition may be recognized by the routine use of the serum or peritoneal amylase. The fact that most cases of traumatic involvement of the pancreas are diagnosed by exploratory laparotomy emphasizes the need to keep this condition in mind when evaluating the patient following upper abdominal injury.

SUMMARY

1. The delayed onset of acute abdominal signs following appropriate trauma should strongly suggest the possibility of traumatic pancreatitis.

2. Establishment or confirmation of the diagnosis by serum or peritoneal amylase determinations is vital since conservative therapy affords the lowest mortality in pancreatitis regardless of type.

3. Uncomplicated cases are best treated by splanchnic blocks for pain relief, gastric suction with nothing by mouth, and the judicious use of parenteral fluids.

4. Associated abdominal injuries such as a ruptured hollow viscus or late complications in the form of pesudocysts require surgical intervention.

5. More frequent use of the amylase test in upper abdominal injury may disclose many otherwise unsuspected cases.

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RAPID LOSS OF PLASMA PROTEIN IN ACUTE SURGICAL CONDITIONS

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THE term hypoproteinemia, taken literally, is quite limited in connotation, and the concept implied, all too often, is correspondingly limited in scope. Many physicians seem to be unaware, or only vaguely aware, of the larger metabolic problem of which plasma protein concentration is only a dependent part.

Figure 1 is a simplified, perhaps oversimplified, representation of a complex physiological mechanism. In health, plasma protein is in a state of dynamic equilibrium. Alimentary protein intake is variable, although for man there is an upper limit of about 150 Gm. a day, the excess being excreted in feces. Usable protein in the small intestine, digested into amino acids, is absorbed. Some of this material is deaminized, is used for energy purposes, and is excreted mainly as urea; some goes to build and nourish body tissues; and some is believed to enter hypothetical "stores" or "pools" or "reserves" of labile protein. Plasma protein is normally derived from tissue proteins and protein stores. In the interest of simplicity, the special role of the liver parenchyma in the manufacture and storage of protein is not indicated here. There is probably interchange between these compartments, as indicated by arrows in the figure. There is also a variable amount of direct loss of plasma protein from breakdown into smaller molecules and secretions into the alimentary tract.

As the diagram suggests, protein depots differ in size. According to Elman,[1] every gram of plasma albumin gained or lost represents roughly 30 Gm. of body protein gained or lost.

To a remarkable degree, all of these variables of gain and loss conspire to maintain in health a constant optimum level of protein in plasma. The mechanism which brings about and controls this equilibrium, which attempts to correct imbalances when they occur, is something we know very little about. But certain it is that a relatively stable concentration of protein in blood and a relatively con-

stant absolute quantity of protein in plasma are requisites of a normal plasma volume and an efficient circulation.
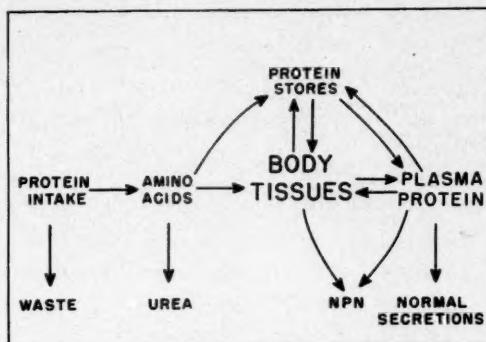
Fig. 1. Schematic representation of the physiological mechanism which maintains plasma proteins in dynamic equilibrium.

The dynamic equilibrium which we are considering is a delicate one, however. There are demonstrable daily variations, but they are within normal limits, are readily compensated for, and do not affect the circulation adversely. But under conditions of trauma or disease, the equilibrium may be quickly and profoundly disturbed. It is important to remember that when the protein of blood is reduced, the whole protein metabolism of the body is affected also, and, conversely, when body proteins are deficient, or a negative nitrogen balance exists, plasma proteins are affected. It should be kept in mind also that the correction of blood deficits by the body, even the transfer of protein from protein stores to plasma, is a relatively slow process, and that restitution of lost plasma protein by intravenous therapy cannot be fully accomplished unless protein deficits in the body as a whole are corrected also.

When sudden loss of protein occurs, plasma protein mass is the first to suffer; the effect on concentration of plasma protein is delayed. In cases of chronic protein depletion, the whole mechanism may be stabilized at an abnormally low level characterized by persistent reduced concentration of protein in peripheral blood. That is the familiar clinical condition generally recognized as "hypoproteinemia." But it is in the acute conditions, associated with rapid losses of plasma protein, before reduction of protein mass is reflected in low protein concentration, that dangerous disorders of physiology may go unrecognized and uncorrected.

During the past 10 or more years, hypoproteinemia has been the object of a great deal of study by Whipple, Cowgill, Weech, Rav-

din, Elman, and numerous other investigators whose writings have contributed materially to our understanding of the subject. The purpose of this paper is not to consider the problem as a whole, but to call attention in somewhat general terms to its most acute phase —the large rapid losses of plasma protein that sometimes occur in suddenly ill or severely traumatized individuals. From our experimental and clinical studies of this phenomenon, my associates and I have come to believe that significant losses of plasma protein are more common in acute surgical conditions than has been generally recognized, and that correction of those deficiencies is a matter of therapeutic importance.

EXAMPLES OF ACUTE PROTEIN LOSS

Acute massive hemorrhages, external or hidden, obviously cause the sudden loss of large amounts of plasma protein. Figure 2 illustrates the problem. Blood samples taken within a few hours revealed only slight changes in hematocrit and plasma protein con-
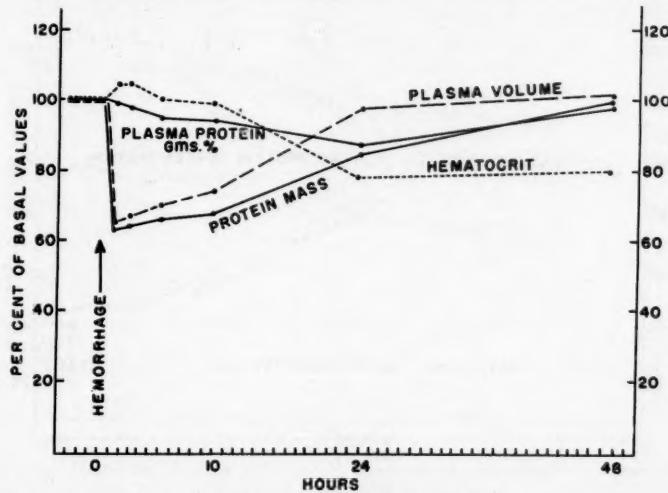
Fig. 2. Effects of a sublethal hemorrhage with spontaneous recovery in a previously healthy dog.

centration, not sufficient in themselves to cause apprehension, but actually the absolute volume of plasma protein was greatly reduced and the circulation was dangerously depressed. The plasma volume returned to normal after 24 hours but plasma proteins were not fully restored for 48 hours. That is something worth pondering. This animal was well nourished and apparently in excellent condition prior to the hemorrhage; his protein stores should have been

well filled; he was permitted to eat and drink as desired following the hemorrhage; yet it required two days for sufficient protein to be manufactured or to be transferred from protein stores to make up the deficit.

The spontaneous slow dilution of blood following hemorrhage has led some writers to conclude that additional plasma is lost; and, in order to explain that supposed loss, abnormal capillary permeability has been postulated. That surmise is incorrect. In uncomplicated posthemorrhagic shock, the circulating blood is diluted by gradual transfer of intracellular water to the vascular compartment, but there is no reduction of total plasma protein mass from generalized capillary leakage.[2]

Exudation of plasma. There are a number of diseases which have in common extravascular escape of large amounts of plasma or serum. Familiar clinical examples include extensive soft tissue injury, intestinal trauma, acute intestinal obstruction, strangulated
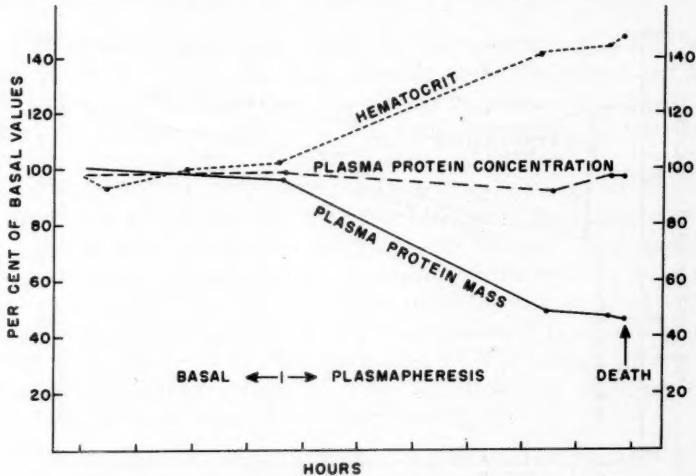
Fig. 3. Effects of rapid controlled removal of plasma from the blood stream. Average results in 21 healthy dogs.

hernia, tortion of viscera, chemical peritonitis, and severe burns. In such cases, there are irretrievable acute losses of protein from the circulation. Effects of such losses on circulating blood may be demonstrated by means of experimental plasmapheresis (fig. 3). The hematocrit rises sharply, as would be expected, but the concentration of plasma protein does not change significantly. Those findings in a patient would hardly lead to a diagnosis of hypopro-

teinemia. Yet the loss of protein is actually very large. In this animal it led to failure of the circulation and death.

When the various diseases mentioned above are reproduced in the laboratory, large amounts of the lost protein are found in and around the injured tissues or organs, but there is no evidence of abnormal capillary permeability to protein in the body elsewhere. In all of these conditions, as in acute hemorrhage, restoration of normal amounts of protein in the circulation by natural processes is a slow process, so intravenous instillation of protein may be helpful and even lifesaving.

Acute infections and extensive inflammatory lesions produce a great variety of pathological pictures, but they have in common well defined areas where capillaries have become abnormally permeable to protein molecules. Seepage of protein into these inflammatory foci in the course of hours or days may be very large indeed. Such
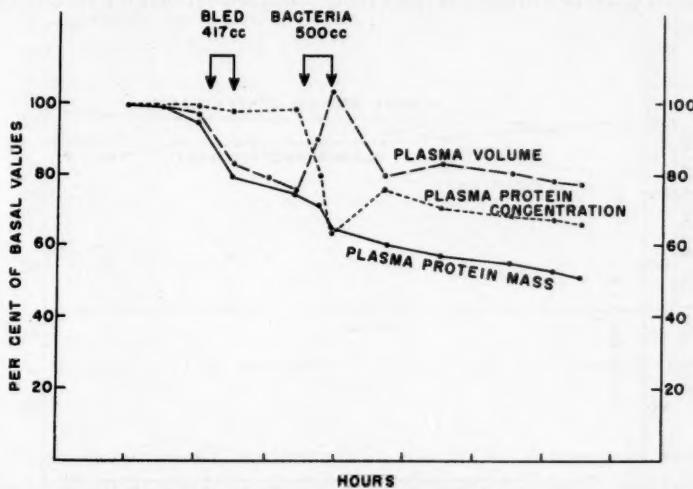
Fig. 4. Effects of bleeding followed by injection of physiological salt solution containing E. coli and S. aureus.

lesions as cellulitis of the leg, lobar pneumonia, acute empyema, or general peritonitis may result in extravascular accumulation of as much as 200 or more Gm. of protein. Cole and Elman state that in severe toxemias "excessive loss of tissue protein, including plasma protein, may occur because of the phenomenon known as 'toxic destruction of protein,' which may lead to the destruction of as much as 250 Gm. of protein a day.[3] Figure 4 illustrates a very acute experiment done to show the effects of injecting several billion pathogenic bacteria into the blood stream of an animal that had already

been bled to reduce its resistance to infection. In less than five hours after the induced bacteremia, plasma protein mass had decreased from approximately three fourths to one half of the normal amount.

Acute dehydration, strange to say, is usually associated with disturbances of protein as well as water and electrolytes. Many physicians are not aware of that. Figure 5 shows the effects of simple narcosis and water deprivation for eight hours in an experimental animal. The plasma protein concentration remained unchanged, but plasma volume decreased appreciably and the plasma protein mass to about 90 per cent of normal. Figure 6 shows the more dramatic effects of acute severe dehydration. This dog received saturated sodium sulfate solution by stomach tube with resultant profuse watery diarrhea. Samples of venous blood showed higher-than-normal protein concentration, but actually there was severe plasma protein deficiency. This deficiency together with greatly reduced plasma volume, undoubtedly contributed largely to circula-
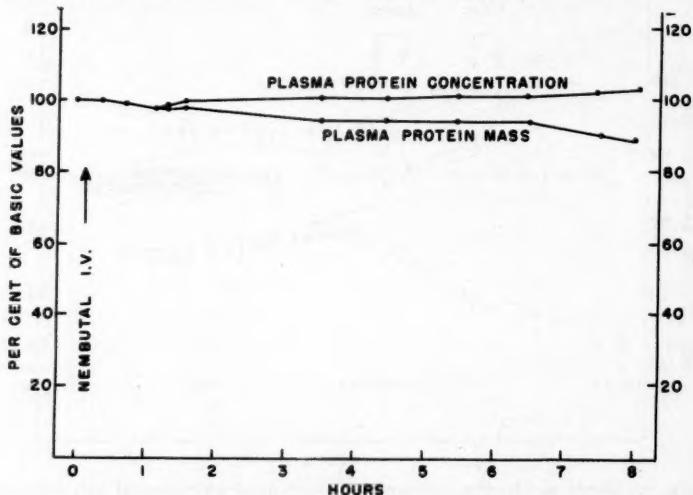
Fig. 5. Effects of simple narcosis and withholding water and food for 8 hours. (Dog 372.)

tory failure and death in this animal. So the treatment of acute dehydration may be even more complex than has been thought. In experiments of this sort we have found therapy more successful, as shown by lower mortality rate and more rapid and complete return to normal, when protein as well as water and electrolytes are injected in order to correct the existing imbalances.

The reasons for reduction of plasma protein mass in acute de-

hydration are not entirely clear. Simple removal of plasma water tends to produce a reciprocal rise in protein concentration, but this hyperproteinemia appears to be resisted by the body. If water is not available to correct the condition by dilution, then enough protein disappears spontaneously from the vascular bed to bring the plasma protein concentration down to within normal limits. This removal occurs without demonstrable change in capillary permeability. We think that some of the "lost" protein is broken down, and that that helps to explain the characteristic rise in non-protein nitrogen.
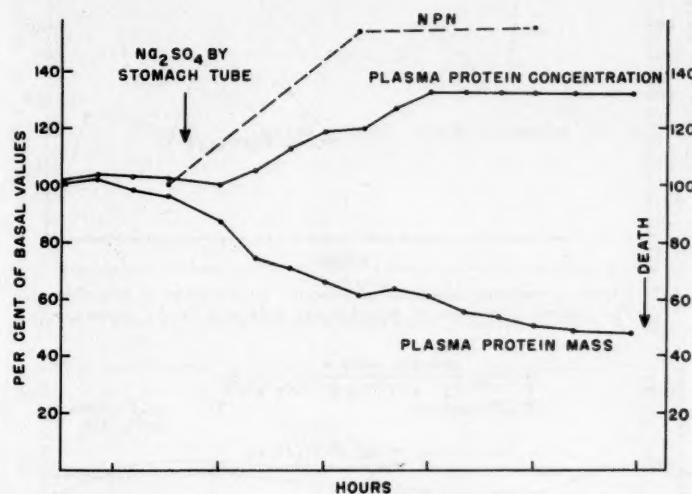
Fig. 6. Effects of acute dehydration produced by means of severe diarrhea. (Dog 610.)

Fractures of long bones have been shown by Cuthbertson[4] and by Howard[5] to be associated with acceleration of protein catabolism and a negative nitrogen balance which persists even in the face of large increases in protein intake. Patients undergoing *major operations,* in which there is considerable loss of blood, as well as inevitable, persistent escape of protein into traumatized and inflammatory tissues, are almost sure to develop hypoproteinemia unless the possibility is anticipated and adequate supportive therapy is administered.

CONDITIONS NOT ASSOCIATED WITH ACUTE PROTEIN LOSS

It must not be thought, however, that all acute diseases or sudden disturbances of vital functions result in significant losses of plasma

proteins. For example, extensive vasodilatation produces a type of "shock" or peripheral vascular failure characterized by extreme hypotension, psychic symptoms, and urinary suppression. Figure 7
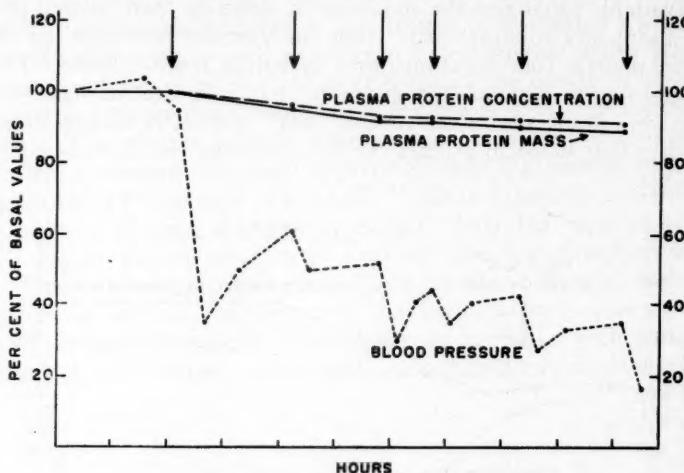
Fig. 7. Effects of repeated injections of procaine intraspinally in a healthy dog. The number and times of injection are indicated by the arrows.
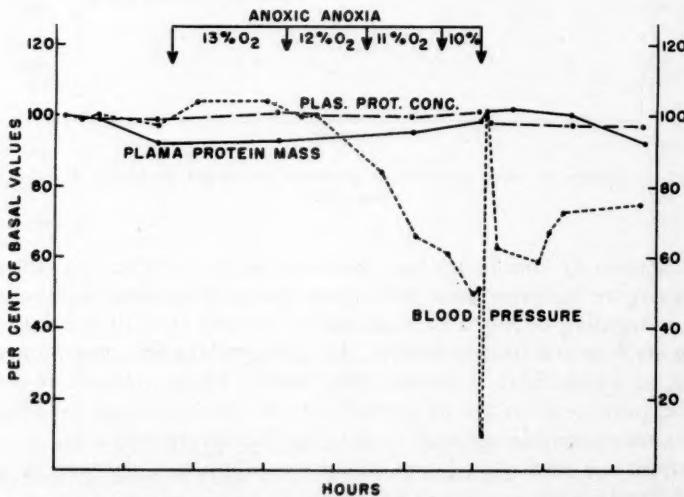
Fig. 8. Effects of increasingly severe anoxic anoxia on circulating plasma proteins. (Dog 67.)

represents an instance of this sort produced experimentally by repeated intraspinal injections of procain. It can be seen that there

was slight dilution of protein, but no more reduction of the total protein mass than would be expected from simple stoppage of all fluid intake.
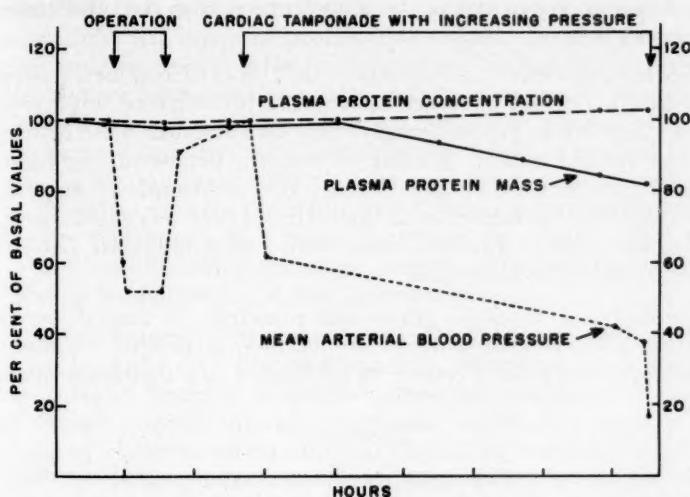
Fig. 9. Effects of cardiac tamponade produced experimentally in a dog.
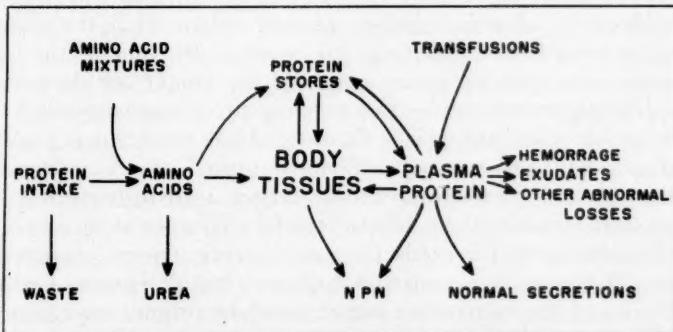
Fig. 10. A schema which indicates some of the variable factors that complicate restoration of deficient plasma protein.

Another sort of physiologic disturbance which may be dangerous or even fatal is due primarily to oxygen lack. The animal represented in figure 8 was subjected to several hours of anoxic anoxia of increasing severity until the circulation suddenly failed and death seemed imminent. The belief is still held by many physiologists and clinicians that anoxia *per se* causes capillaries to become abnormally

permeable to proteins. Within limits of anoxia ordinarily encountered, we have not been able to discover in our experiments with dogs any support for that belief. Certainly in this instance there was no significant change in the concentration or in the total amount of plasma protein.

We have not made an exhaustive study of cardiac injury, dysfunction or disease, but in so far as we have investigated those conditions, clinically or experimentally, they do not cause acute losses of plasma protein except in cases of sudden, persistent, high-grade venous back-pressure. Figure 9 illustrates an instance of acute cardiac tamponade produced experimentally. There was slight increase in protein concentration associated with a slow moderate reduction in total plasma protein mass.

Similarly, in shocklike states due primarily to central nervous system injury, peripheral nerve stimulation, or psychic trauma, we have not found plasma protein to be affected to a significant degree.

COMMENT

In all of the pathological conditions, experimental or clinical, which have been mentioned, in which there are acute losses of plasma protein from the vascular bed, the obvious rational treatment is to restore those losses by intravenous injections of plasma, whole blood, or albumin solution. If that is done while the plasma protein is being lost, or within a few hours of the time of the loss, full restoration may be anticipated; but the longer the delay, the more difficult or impossible it is to bring the plasma protein back to normal. It is well recognized that after acute hemorrhage prompt transfusion will restore blood volume to normal, whereas after the development of pronounced shock, larger transfusions may be needed, and even they may be unsuccessful. It is not so well known that the same principle holds for acute severe plasma protein deficiency. We have discovered that dogs with lethal degrees of crushing injuries of the extremities can be saved by prompt injections of large amounts of salt solution and plasma, but that if treatment is delayed for many hours, similar or even larger injections will not be successful.[6]

A different situation obtains, however, when acute protein losses occur in individuals who have long been ill, whose protein reserves are already low. If, for any reason, protein intake has been deficient for a long time, or if protein losses have been excessive, the amount of body tissue is reduced, available protein stores are greatly diminished, and plasma proteins fall far below optimum levels. Whipple[7] and his associates found that fasting alone used up body

proteins and reduced plasma protein mass by as much as half. Liver disease may so disrupt the whole mechanism of nitrogen metabolism that subnormal levels of protein are produced and maintained in the body and in plasma. Injury or disease may cause such persistent abnormal losses of protein that tissues and plasma are depleted and protein stores become exhausted. So familiar is this situation that we have come to expect patients who are chronically ill, or people who are malnourished, to have low plasma proteins. They become stabilized at abnormally low levels of protein physiology. They are poor medical risks, and poor surgical risks. If they should be subjected suddenly to additional acute protein losses, say from pneumonia or a major operation, the results might well be disastrous. Should that happen, prompt administration of blood or plasma is indicated, and may be imperative.

Effective treatment of these chronically ill, acutely depleted patients is far from easy, however (fig. 10). When protein is injected intravenously, much of it quickly leaves the vascular bed. Some may escape through injured or inflamed capillaries; some is destroyed by ordinary or extraordinary catabolic processes; much of it goes to fill the relatively enormous needs of the body tissues for proteins. If there is time and opportunity, a high-protein diet may and should be used to augment parenteral protein alimentation. It appears that abnormally low levels of protein in plasma can be permanently raised only to the degree that the protein needs of the body as a whole are satisfied. An important rule of therapy is that during the period of protein build-up the caloric requirements of the body must be met fully, otherwise much of the injected protein will be broken down and be used for energy purposes. All the while, care must be taken not to load these patients with more water or electrolytes than they can handle. Whipple has stated that at least 2,500 cc. of plasma may be needed to raise plasma protein concentration in hypoproteinemic patients. Allen and associates[8] were able to correct plasma protein deficiencies in 9 acutely ill patients, who had previously developed hypoproteinemia and anemia, by injection of 500 to 1,500 cc. of plasma and blood daily for one or two weeks. Elman[9] has demonstrated the value of oral and intravenous administration of amino acids. In less urgent cases, the oral route is often the simplest, safest, and most successful way of supplying needed protein.

Some chance observations made during the course of our study have interested and puzzled us a great deal. One was that intravenously injected protein which leaves the vascular bed cannot be quickly recovered by the blood even in the presence of urgent need. For example, if a normal dog is given a transfusion, the excess

plasma protein will disappear from the blood stream within 2 or 3 hours. If the animal is then bled rapidly so as to produce an acute plasma protein deficiency, needed protein is not transferred back into the blood any more rapidly than in the control animal which had not received a fortifying transfusion.

Another observation was that, in the presence of acute plasma protein deficiency, water by mouth tends to increase somewhat the absolute amount of intravascular protein, whereas water given intravenously, in the form of salt or glucose solution, tends to reduce further the already deficient amount of circulating plasma protein.

Summary

Hypoproteinemia should be viewed as a manifestation of a general disturbance of protein metabolism. In health, plasma proteins are maintained in dynamic equilibrium, but in conditions of severe trauma or disease the equilibrium may be upset with adverse effects upon the circulation.

Long-standing protein deficiencies are easily recognized by the subnormal concentrations of protein in peripheral blood, but in cases of acute protein loss, hypoproteinemia is more difficult to detect, inasmuch as the total plasma protein mass may be seriously depleted without commensurate reduction in plasma protein concentration.

Many serious surgical conditions are associated with rapid, large losses of plasma protein. These losses may be dangerous. They should be recognized and corrected. Better still, they should be anticipated and counteracted. These losses of protein do not necessarily coincide with or parallel fluid and electrolyte imbalances.

The problem of treatment involves not only replacement of lost plasma protein, but also correction of the associated abnormal protein metabolism and satisfaction of the protein demands of the body generally.

Correction of plasma protein deficiencies by natural processes is a relatively slow process, hence the rational of parenteral administration of protein. In acute deficiencies, prompt injection of protein (in the form of whole blood, plasma, or albumin solution) is more successful than delayed treatment.

In chronic hypoproteinemia, large amounts of protein will be needed, combined with an alimentary and parenteral regimen which fully meets the caloric requirements of the body. A particularly difficult therapeutic problem is presented by patients with well

established hypoproteinemia who are suddenly subjected to additional acute plasma protein losses.

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A LONG-LASTING LOCAL ANESTHETIC SOLUTION— USE IN THORACIC SURGERY

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RECENTLY a new local anesthetic solution, Efocaine (Fougera), has been marketed. The active ingredients of the solution are procaine (1 per cent), procaine hydrochloride (0.25 per cent), and butyl-p-aminobenzoate (5 per cent). The solvent is a mixture of polyethylene glycol-300 (2 per cent) and propylene glycol (78 per cent), with sodium metabisulfite (0.1 per cent) and phenyl mercuric borate (1:25,000) added for preservative action.

Efocaine is a clear non-oily solution. The anesthetic base is completely soluble in the solvent, but only slightly soluble in water. Thus, with the solution at a critical saturation level, the anesthetic base is precipitated out when tissue fluids come in contact with the solution. The crystalline anesthetic depot that forms is slowly absorbed over a period of 6 to 12 days, exerting a local anesthetic effect continuously. Weinberg,[1] in an exhaustive study of the action of Efocaine on nerves, muscles, skin, and subcutaneous tissue, was unable to find any deleterious effect, either immediate or delayed. Other investigators[2-5] have recently reported clinical studies demonstrating the efficacy of Efocaine in the management of postoperative pain. Therefore, a trial of Efocaine in thoracic surgery seemed indicated, as the advantages of a prolonged intercostal nerve block following thoracotomy are many, subjectively and objectively. Without pain the patient rests more comfortably, and has an earlier return of the desire to get well. Appetite, fluid intake, and elimination quickly return to a normal level. The ability to cough without exquisite pain allows thorough cleansing of the tracheobronchial tree by expulsive coughing, thus avoiding obstructive atelectasis. The use of an analgesic, particularly morphine, to suppress pain is undesirable as it depresses the cough reflex. Further, if pain is prevented by nerve block, the patient will turn in bed more readily and breathe deeply, thus helping to avoid atelectasis due to poor aeration.

TECHNIC OF USE

The easiest and most accurate method of injecting Efocaine is through the open chest at the time of thoracotomy, when, under

direct vision, the intercostal nerves can be injected with 100 per cent accuracy. Only 1 cc. of the solution is required, per nerve, to insure adequate prolonged anesthesia. Usually five nerves will need to be blocked; the one corresponding to the rib removed, along with the two immediately above and below. It is important to avoid injecting the solution under the periostium, for then the nerve is not affected. Intravenous injection should be avoided, although in rabbits 0.6 cc. has been injected intravenously without ill effects.

When it is desirable to use Efocaine other than at thoracotomy, the usual technic of intercostal block is used. Procaine is used to obtain skin anesthesia, and after the needle tip is in the vicinity of the nerve, about 1 cc. of procaine should be injected before an equal amount of Efocaine. Otherwise, there is a rather severe, but short-lasting, burning pain at the site of the injection.

To avoid allergic reactions, an intradermal sensitivity test should be performed with 1 minim of Efocaine. A positive reaction is a contraindication to the use of the solution.
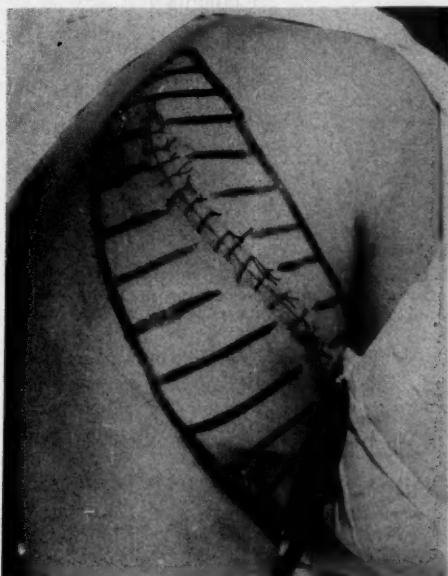
Fig. 1. Case 1. Marked area indicates anesthesia existing 48 hours after inter-costal block was performed.

The following case reports illustrate the use of Efocaine in thoracic surgery:

CASE 1. A 27 year old woman with recurrent episodes of hemoptysis. Chest

roentgenogram was negative, bronchogram showed right middle lobe bronchiectasis. The affected lobe was resected through the bed of the seventh rib posteriorly. The fifth through the ninth intercostal nerves were injected with 1 cc. of Efocaine each prior to closure of the chest. Recovery was excellent, and she was discharged on the ninth postoperative day. Only 10 mg. of morphine were required for pain relief the day of operation, with a similar amount being required for the first postoperative day. Thereafter her discomfort was easily controlled with aspirin. Figure 1 demonstrates the zone of anesthesia on the second postoperative day.
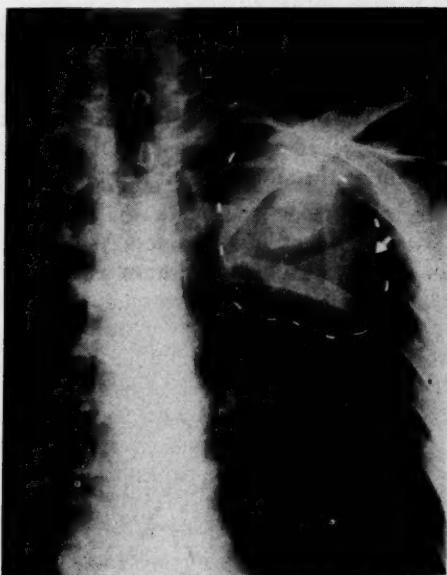
Fig. 2. Case 2. Roentgenogram demonstrating tumor mass (dotted outline) in the upper left thorax, with erosion of the fourth rib (note end of rib at arrow).

CASE 2. A 45 year old man with a malignant tumor involving the posterior superior portion of the left chest wall (fig. 2). Severe pain for several weeks had been forcing him to take several grains of codeine daily, and about one-half pint of whiskey per night for sleep. On the day of admission the second through the fifth intercostal nerves on the left were blocked posteriorly with complete relief of all pain. Figure 3 demonstrates the zone of anesthesia 48 hours after the block was performed. At operation the tumor mass, including portions of the second through the sixth ribs, was resected. The corresponding nerves were cut, thus obviating the use of Efocaine further.

CASE 3. A 29 year old man with chronic empyema on the left. At operation the peel was found to cover the entire surface of the lung, except for the mediastinal surface. Decortication was performed. The fifth through the ninth nerves were injected (the seventh rib had been resected). He required 300 mg. of demerol the day of operation and 400 mg. the following day. As he had good skin anesthesia, he was given only aspirin thereafter, and this completely relieved all pain. Undoubtedly the edema of the chest wall, which occurs following decortication, affected nerves other than those blocked.

CASE 4. A 56 year old woman with far advanced cancer of the right lung. Exploratory thoracotomy (diagnosis not made preoperatively) through the bed of the fourth rib anteriorly revealed complete collapse of the middle and lower lobes, with seeding of the tumor onto the mediastinal structures and the diaphragm. Pneumonectomy was performed for palliation. The second through the sixth nerves were injected with 1 cc. of Efocaine each. She required 75 mg. of demerol the day of operation, 10 mg. of morphine (demerol produced nausea) on the first postoperative day. Recovery was uneventful; excellent skin anesthesia was present at the time of discharge.
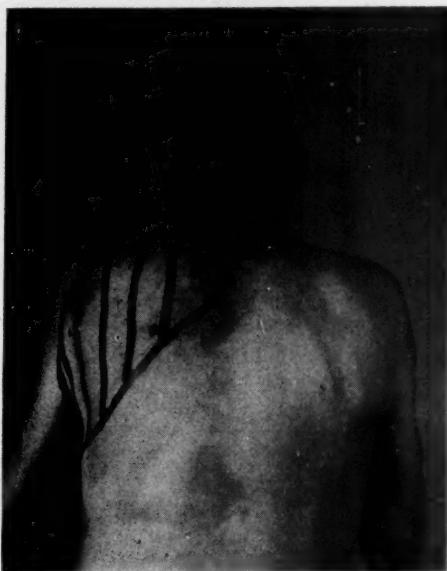
Fig. 3. Case 2. Marked area illustrates zone of anesthesia 48 hours after intercostal block. Dots posteriorly indicate sites of injection (a pigmented nevus is present within the zone of anesthesia, and resembles a dot).

CASE 5. A 66 year old woman with cancer of the middle third of the esophagus. The chest was entered through the bed of the eighth rib; examination showed the lesion to be resectable. Therefore a portion of the fifth rib was also resected through the same skin incision, and the entire thoracic esophagus, except for a 3 cm. segment in the apex of the chest, was resected, with performance of an esophagogastric anastomosis. The second through the tenth intercostal nerves were injected with 1 cc. each of Efocaine. The postoperative course was extremely benign. She required 200 to 300 mg. of demerol daily for the first four days, although skin anesthesia was complete. It is therefore believed that visceral pain may have occasioned her discomfort.

SUMMARY

The composition, mode of action, and technic of use of Efocaine, a long-lasting local anesthetic, are given, with case reports illustrat-

ing its use in thoracic surgery. The amount of analgesic drugs needed to control pain was found to be significantly reduced.

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THE CASE AGAINST TREATING ULCERATING LESIONS OF THE STOMACH MEDICALLY

The universally poor results following surgery for carcinoma of the stomach are well known. With present methods at our disposal any appreciable improvement in this situation will come about in two days: First, earlier diagnosis with immediate surgery and, second, removal of lesions that are known to be precursors of malignancy. More radical removal of the stomach including more frequent use of total gastrectomy and wider removal of probably involved surrounding structures may add to the length of survival of some individuals, but I believe the increased salvage rate will, indeed, be small. Total gastrectomy, no doubt, is indicated for those individuals in whom the lesion, so far as the surgeon is able to determine at the time of the operation, has not extended beyond resectable limits and in whom a less radical operation obviously does not permit adequate removal of the gastric wall beyond the local lesion. Microscopic study of the removed specimen has frequently shown that a sufficient amount of both gastric and duodenal wall has not been accomplished. The extirpation of an entire organ involved with malignancy follows the principles of good cancer

surgery and, indeed, would be desirable in all cases of cancer of the stomach, weer it not for the fact that many such individuals are left gastric cripples. The mortality following total gastrectomy in the hands of competent surgeons has been reduced to an acceptable point considering the serious nature of the disease for which radical surgery is done. I seriously doubt that the morbidity rate is acceptable in most cases considering the improved end results accomplished by such a procedure.

The acceptance by medical men of any teaching at variance to that of their predecessors is, indeed, a slow evolution. It took many years to convince surgeons that gastroenterostomy is an operation with very limited indications in the treatment of gastroduodenal lesions. Early ambulation in the postoperative care of patients is a striking example of a more recently changed attitude. It is high time that we revise our attitude toward the treatment of gastric ulcer and realize that there is no place for the medical treatment of such a lesion in an individual who is a good surgical risk. It is unfortunate that any ulcerating lesion of the stomach is ever referred to as being a peptic ulcer. For to do so is to give the impression that the lesion is benign. No lesion of this nature should ever be considered to be benign until an adequate examination by a competent pathologist has been made by microscopic examination of the ulcer crater and surrounding lymph nodes, regardless of the age of the patient or the duration of the ulcer.

In Hawaii, where the population is approximately 40 per cent Japanese and in whom we have found ulcerating lesions of the stomach to represent malignancy more than twice as frequently as in any other race of people, we have long since become acutely aware of the dangers of treating any such ulcer by conservative methods. There are many reasons why all good-risk individuals with ulcerating lesions of the stomach should be subjected to operation and I do not believe anyone can advance any logical reason why they should be treated medically after such a diagnosis has been made. First and foremost, there is no way of differentiating between a benign and a malignant ulcer of the stomach in its early stages. No surgeon will have any appreciable number of cures and very few five-year survivals, even by the most radical type of surgery, in individuals in whom the diagnosis of cancer of the stomach is made clinically.

In my own series of cases only 25 per cent of those who survived resection lived for as long as five years, and in none of these was there demonstrable lymph node involvement by malignancy at the time of operation. It is impossible to accurately determine how

many gastric ulcers, benign in their incipiency, ultmately undergo malignant degeneration. From clinical observations alone, it would seem that the number is far greater than we have been led to believe. Many individuals with gastric carcinoma give a history of gastric symptoms of many years' duration compatible with a diagnosis of a benign lesion of the stomach. A number of acute and chronic ulcerating lesions of the stomach in various age groups and races have been resected which we thought were clinically benign and on careful microscopic study of the ulcer crater, malignant changes have been found. One individual with a small ulcer crater in the lesser curvature of the stomach with a normal gastric analysis refused surgery and five years later gastric analysis revealed no free hydrochloric acid. At operation a large mass with extensive metastases which was found in the lesser curvature of the stomach made the lesion entirely irremovable. While these experiences do not prove that cancer developed in a previously benign lesion, juries have sentenced men to hang on circumstantial evidence of a less convincing nature.

Many medical men and a high percentage of surgeons with whom this problem has been discussed believe in giving the individuals wit han ulcerating lesion of the stomach a period of medical treatment before subjecting them to surgery. No doubt a number of these lesions will clear up, some permanently, some temporarily to recur again and again, and some apparently temporarily even though there is an underlying malignancy. Why spend this time temporizing when this may well represent the period during which malignant cells spread beyond points of resectability? If it is dangerous, as we all believe, and as we impress upon medical students and house staff to palpate indiscriminately a malignant lesion of the breast, lest we dislodge malignant cells into the lymphatic or vascular spaces, how much more likely and how much ealier in the course of this disease is this likely to occur in the wall of the stomach where the lesion is undergoing more or less constant massage by gastric peristalsis.

It has been suggested that all patients over 40 years of age who have chronic recurring gastric ulcers should be operated upon and that patients under 40 years of age who have a short history of "peptic" ulcer and who on roentgenologic examination of the stomach appear to have small chronic ulcers should have the benefit of a trial of medical treatment because many such ulcers will heal. It is further contended that any patient with gastric ulcer not operated upon should have repeated roentgenologic examinations at three-month intervals for an indefinite period of time. It seems to me that this method of handling such cases largely defeats the pur-

pose for which it is intended. Since it is so important to remove these lesions when they are malignant at the earliest possible moment this opportunity is lost, and by this method of procedure a possible precancerous lesion is allowed to progress until it shows evidence of malignancy before it is removed. Malignancy of the stomach in patients under the age of 40 occurs with sufficient frequency in our experience to make this method of handling such cases a dangerous procedure. Just how many gastric ulcers thought to be benign by all methods of examinations at present at our disposal —including roentgenologic investigation, gastroscopic examination, gastric analysis and search for malignant cells in gastric washings, that later prove to be malignant—one cannot estimate with any degree of accuracy. In the experience of some writers who have large series of cases from which to draw conclusions, it is thought to range between 10 and 20 per cent, certainly a number far in excess of what the mortality would have been, had all of these cases been sujcedted to gastric resection. Some seem to forget that the ultimate mortality for unresected carcinoma of the stomach is 100 per cent.

To me at least the case of resecting all ulcerating gastric lesions could rest at this point with the firm assurance that it is based on sound surgical principles not only by the experiences we have had, but by experiences that have accumulated in all clinics that are interested in this problem. To those skeptical internists and to the diminishing number of our surgical colleagues who are yet to be convinced, additional reasons may be enumerated.

All of us, I am sure, can recall a number of instances of chronically disabled individuals who have been restored to good health and a useful career following resection of a chronic ulcerating lesion of the stomach and many have asked the embarrassing question, "Why was this not done years before?" A discussion of hemorrhage from ulcerating lesions of the upper gastrointestinal tract continues to appear at frequent intervals on the programs of most medical gatherings and many articles in current medical literature are devoted to this subject. The importance of these episodes and the unsatisfactory approach to the problem is attested to by the importance given to the subject and the lack of unanimity of opinion regarding how they should best be handled. Regardless of the method used in their treatment, a considerable number of these cases terminate fatally, and a considerable number are the result of ulcerating lesions of the stomach. At times ulcerating lesions of the stomach do perforate and though the outcome of such catastrophies are less to be feared now than in previous years, they are still not without some mortality regardless of the method used in

their correction. Needless to say, had these lesions been removed at a more opportune time, for reasons previously mentioned, the results would have been correspondingly better.

It is difficult for surgeons to keep abreast with advances being made that enable them to surround their patients with greater safety measures during and subsequent to operative procedures. How much more difficult is it for the internist, always a bit suspicious of his surgical colleagues, to begin with, to realize to what extent these advancements may have affected his time-honored methods of therapy.

In competent hands gastric resection for the usual ulcerating lesion of the stomach, when doubt exists as to its benignancy or malignancy, carries with it a very low mortality rate. The difficulties encountered so frequently in coping with ulcerating lesions of the duodenum are rarely met with in similar conditions in the stomach. Stomal ulcers following gastroenterostomy or gastric resection for duodenal ulcers has been the complication most to be feared and the principal stimulus for popularizing and defending the use of vagotomy. While, no doubt, stomal ulcers do follow gastric resections for ulcerating lesions of the stomach, they must be exceedingly rare. I have never seen such a complication regardless of the extent of the stomach removed, and many other writers on the subject record the same observations. In our experience and in the experience of many surgeons with a far greater number of cases from which to dra wconclusions, the results of gastric resection for benign lesions of the stomach continue to be one of the most gratifying chapters of surgery.

Does anyone in these days of enlightenment advise watching a lesion of the breast to see whether or not it will ultimately become malignant? Do not thoracic surgeons urge the investigation of all lesions of the lung in which doubt exists as to their nature? Why, then, should we as abdominal surgeons condone the archaic teaching that ulcerating lesions of the stomach are to be considered as benign until giving evidence to the contrary?

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BOOK REVIEWS

THE CLINICAL USE OF FLUID AND ELECTROLYTE. By JOHN H. BLAND, Asst. Professor of Medicine, University of Vermont School of Medicine. Philadelphia and London, W. B. Saunders Company, 1952. 259 pages. Paper binding.

This volume is good in most respects. It dwells in some detail upon the fluid and electrolyte requirements of the body in health and disease. It is more easily read and understood than books such as Gamble's "Extracellular Fluid," yet it lacks some of the experimental work presented there. There are many charts and graphs. All in all, the book is very practically written except for one glaring deficiency—there is no index. Thus, one cannot use this volume as a ready reference but must wade through considerable material to find the desired information. The addition of an index would greatly enhance its value and use.

The author has brought out one good and interesting fact, i.e., that we tend to recognize fluid and electrolyte disturbances in the aged as being "due to his age" rather than what they actually are, and thus we are allowing them to die natural deaths rather than correct their abnormalities and allow them further years of life.

The print is rather small. The illustrations, while occasionally hard to understand (this being usually the reader's fault) are fairly good. The paper is good. The binding will probably last as long as this volume is modern considering the rapidity with which new developments are being worked out. Every hospital should have this volume and it should be required reading of every house officer. All physicians and surgeons would profit by having digested this work.

A. H. LETTON, M. D.

CURRENT THERAPY 1952. Ed. by HOWARD F. CONN. Philadelphia, W. B. Saunders Company, 1952. 847 pages. Cloth binding.

Here is this year's edition of an ever increasingly popular book. This is not an ideal book for a specialist but is designed to act as an emergency aid to therapy.

The treatment in each case is briefly outlined and there is no detail given, which is correct and proper, for it is the hope of the editor and publisher that the physician will use this as a guide to what is thought to be the best therapy and that he will find details elsewhere. When there is a difference of opinion as to therapy, all methods are presented but none are discussed.

This edition finds 189 new contributors swelling the size of the volume with their writings and knowledge. There is much of the book which is not changed but there are many revisions as well as the additions. The print is easily legible. The paper is of good quality and the binding is fair.

This volume should be popular with the general practitioner but only as an outline of what is considered good treatment at present.

A. H. LETTON, M. D.

CALLANDER'S SURGICAL ANATOMY ed. 3. By BARRY J. ANSON, M.A., Ph.D. (Med. S.), Professor of Anatomy, Northwest University School of Medicine, and WALTER G. MADDOCK, M.S., M.D., F.A.C.S; Elcock Professor of Surgery, Northwest University School of Medicine; 929 illus. 1074 pages.

This is a welcome addition to the surgical armamentarium, for the last edition has been out of date too long. The former editions have been worn threadbare by use and, in some instances, while the anatomy remains the same, the requirements of today's surgeon have extended themselves beyond the scope of the old edition.

The new authors, who courageously have picked up the torch, have done an excellent job in revising, rewriting and re-editing this volume. They have done this single-handed for Dr. Callander has left no new manuscript or even an outline for future editions. Thus, one group of architects had to design a new building on old foundations without the help of the former architect's plans.

The print is easily readable. The style is free. The paper is of good quality and the binding will resist the wear which this volume will be required to withstand.

Every operating room library should have a copy.

A. H. LETTON, M. D.

UROLOGICAL PATHOLOGY. By PETER A. HERBUT, M.D., Professor of Pathology, Jefferson Medical College, and Director of Clinical Laboratory, Jefferson Medical College Hospital, Philadelphia, Pa. Pages 1,222 with 527 illustrations, including 2 in color. Philadelphia, Lea and Febiger, 1952.

This is an excellently written and planned text on urologic pathology. It is very easily read. It is well indexed even to the smallest subheadings and can be easily assimilated with a minimum of study.

Each pathologic entity is described adequately and in detail under headings of distribution, cause, clinical manifestations, gross appearance, microscopic appearance, complications, clinical pathologic corrolations, diagnosis, treatment, and prognosis. Taking all these factors into consideration, it makes an excellent reference text for general usage in urologic diseases as well as a good guide to urologic pathology.

The book is quite readable, being nicely printed on glossy paper. The illustrations, while black and white with but two exceptions, plainly illustrate as they are intended to do. The binding appears to be good quality and will probably stand more than the usual usage.

This book would be useful in all medical libraries; for the general practitioner, for the specialist in any field who is interested in urologic pathology, for the medical student, and for the urologist himself. It is a fine reference work in general for conditions of the urologic tract and male genital tract and specifically excellent for its surgical pathologic data.

CURTIS W. BOWMAN, M.D.

ABSTRACTS FROM CURRENT LITERATURE

SURGICAL ASPECTS OF DIVERTICULITIS. Neil W. Swinton. *American Journal of Surgery 82:*603-610 (Nov.) 1951.

Cases of diverticulitis requiring surgical intervention are uncommon. This report is based on a review of the records of 60 patients operated upon for this condition at the Lahey Clinic since 1938. In general, only those patients who have developed complications of the inflammatory condition are operated upon and the usual medical management is applied to the great majority of cases. The author believes that surgical intervention is indicated in those patients with diverticulitis who exhibit evidence of acute perforation, abscess formation, obstruction, fistulas, repeated hemorrhages, a tumor of the left colon in which malignant disease cannot be excluded and those who fail to obtain relief for their symptoms by conservative measures.

The type of surgical procedure indicated will vary with the individual case. In acute perforation, simple closure of the perforation with proximal colostomy is the procedure of choice. In abscess formation, the author feels that incision and drainage followed by resection of the diseased portion of the bowel, after the acute inflammation has subsided, is indicated. For the patient with obstruction, he advocates a three stage procedure: right transverse colostomy for diversion; resection of the tumor; and later closure of the colostomy. He states that, in case of an obstructing tumor when malignancy cannot be ruled out, segmental resection of the involved area with examination of the excised tissue must be carried out before a final decision is made as to the operative procedure to be used. In case of fistulous tracts complicating the disease, the three stage procedure is generally used.

A brief discussion of the differential diagnosis of the disease is included.

R. H. S.

PROCTALGIA FUGAX. Joseph D. Karras and Gaspar Angelo. *American Journal of Surgery 82:*616-625 (Nov.) 1951.

"Proctalgia fugax is a well defined symptom complex of obscure etiology and pathology which is characterized by sudden and irregularly intermittent attacks of intense pain of short duration occurring in the region of the internal sphincter and anorectal ring."

In this paper, the authors call attention to this entity of obscure etiology which is devoid of related objective findings and for which they suggest no therapy more definitive than antispasmodics. Twelve cases of rather varied nature are presented. Nitroglycerine under the tongue seems to abort an attack in some instances.

Among several discussants, Neil W. Swinton offered as a possible explanation of the phenomenon, a case report in which the attacks were relieved by presacral neuronectomy.

R. H. S.

THE TREATMENT OF EDEMA OF THE ARM. William T. Foley. *Surgery, Gynecology and Obstetrics 93:*568-574 (Nov.) 1951.

Herein, the author reports 7 cases of edema of the arm following axillary surgery and 1 case following thrombosis of the axillary vein (post-traumatic)

which have responded to a program of treatment combining gravitational drainage, massage, compression and dehydration. The gravitational drainage was effected by traction suspension on a snug fitting glove while the patient is in bed. This is augmented by compression bandages while the patient is up and about. Self massage for ten minutes twice daily has been augmented by whirlpool baths. At the onset of treatment, a program of dehydration is carried out for one week. This consists of the daily or every other day administration of mercurial diuretics in addition to a 2 Gm. salt diet.

In this small series of cases, marked improvement in swelling, pain, and cosmetic appearance has resulted and has been maintained.

R. H. S.

CARCINOMA OF THE ESOPHAGUS OR CARDIA OF THE STOMACH. Y. K. Wu and H. H. Koucks. *Annals of Surgery 134:*946-956 (Dec.) 1951.

From the Department of Surgery of the Peking Union Medical College comes this report of the analysis of 172 cases of cancer of the esophagus or cardia. The extremely high incidence of this disease in North China is probably related to the dietary habits of the people. The authors consider a 50 per cent incidence of habitual drinking of *pai kan,* a locally produced distilled alcoholic beverage, in this group compared with 12 per cent in a control group to be statistically significant. A positive family history of carcinoma of the esophagus was noted in 22 per cent of the patients.

The resectability rate was 47.1 per cent of the total group and 63.3 per cent of those operated upon. The operative mortality in 81 recent resections was 17.3 per cent and for the 26 resections performed during the year 1949 was 7.7 per cent. There was no death among 13 patients subjected to resection in the first five months of 1951.

Forty-one of the 67 patients who survived resection are living from a few months to more than three years after operation. Twenty-four others have died from recurrence of the disease. Of 12 patients who were subjected to resection in 1940 and 1941, 1 lived for five years and another was still living and well at the time of this report. From this, the conclusion is drawn that resection is definitely worth while in those cases in which the lesion has not yet spread widely but that palliative resection, when it is not possible to resect the gross tumor completely, is not advisable.

The authors emphasize the importance of recognition of the early symptom of intermittent obstruction to the swallowing of food and plead for an extremely alert attitude toward any patient of cancer age, especially a male, who mentions vague difficulty in swallowing.

R. H. S.

THE GALLBLADDER. Comparison of Roentgen and Pathological Findings. P. E. Russo and C. J. Cavanaugh. *Southern Medical Journal 45:*232-234 (March) 1952.

This presentation, a continuation of a previous report, is an analysis of 1,000 consecutive cholecystograms demonstrating the diagnostic accuracy as proved by surgery. Of the 1,000 examinations, 635 were interpreted as normal and 365 were pathological. In this series, 130 came to surgery. Fifty-four cases had been diagnosed as having stones (32 opaque and 22 nonopaque) and at operation stones were found in each instance. Seven cases exhibited

poor visualization and all proved to have stones. Of the 65 cases in which no visualization was obtained, 62 had stones at operation, 2 had chronic cholecystitis without stones and the remaining instance was a patient who had a normal gallbladder and an advanced cirrhosis of the liver. Two cases of calcified gallbladder were diagnosed preoperatively and 2 cases diagnosed as having a normal gallbladder exhibited no disease of the organ at operation.

The authors attribute this high degree of accuracy to meticulous adherence to a rigidly standardized technic which they describe in detail and conforms to that now employed in most centers. They have abandoned the fatty meal as being of no value. In 50 of the cases exhibiting nonvisualization of the gallbladder with a standard dose of dye, the procedure was repeated with a double dose and 5 of these, or 10 per cent, then exhibited normal visualization.

R. H. S.

ACUTE APPENDICITIS IN THE AGED. William I. Wolff and Robert Hindman. *Surgery, Gynecology and Obstetrics* 94:239-247 (Feb.) 1952.

Critical consideration of surgical conditions in the aged is compelling, not only because diminished physiologic reserve in this segment of the population make delay and faulty management poorly tolerated, but also because of the numerical increase in the segment of population 60 years of age and over. The margin of tolerable error is narrow in the aged and this is particularly true of those entities usually referred to as the "acute surgical abdomen."

In this consideration of one phase of this problem, the authors have studied the case records of all instances of acute appendicitis in persons 60 years of age and over treated on the surgical wards of the Cornell division of the Bellevue Hospital during the decade of 1940 through 1949. A control group of cases during the same period (those from 30 to 40 years of age) was selected for comparison. There were 88 cases in the 60 plus age group and 274 in the control group. Only pathologically confirmed cases of acute appendicitis were included.

The comparative duration of symptoms in the two groups were studied from several approaches. The inference drawn from these considerations is that the appreciably high incidence of peritonitis in the older age group cannot be ascribed to delay in hospitalization.

Realization of the fact that elderly patients frequently advance surgical pathological conditions with a paucity of signs and symptoms points up the need for a different set of diagnostic standards for this age group. Though pain occurs in nearly all the cases, the abdominal findings conform to no consistent pattern, often varying with the time of examination and the examiner. Tenderness, frequently associated with muscular spasm, was the most constant feature. In an analysis of errors or delay in diagnosis, it was found that serious error or delay occurred in 5.5 per cent of the control group and over four times as often (25 per cent) in the 60 plus age group.

Analysis of the type of incision employed in the surgical treatment further points up the uncertainty of diagnosis.

As might have been expected, the complication rate in the older group was a little over three times that of the control group. The mortality rate in the older group was 4.5 per cent as opposed to 0.7 per cent in the control group.

The authors conclude that appendicitis is a more fulminating disease in the

aged, exhibiting frequent abberations in its clinical manifestation and calls for the exercise of more careful clinical judgment than does the same disease in younger adults.

R. H. S.

OPERATIVE TREATMENT OF FLATFOOT. Ben L. Schoolfield. *Surgery, Gynecology and Obstetrics 94*:136-140 (Feb.) 1952.

Joint fusion operations for the correction of flaccid flatfoot are outmoded and should be abandoned. On the premise that the position of the os calcaneus is the key to the deformity and that the vertical attitude of this bone is normally maintained by a deltoid ligament of normal length, the author advocates shortening of the deltoid ligament. The description of the technic he employs is quoted:

"The operative technic is simple. The incision begins behind the medial malleolus and ranges down and around that bone to curve forward to the talonavicular joint; this allows adequate exposure when the skin is dissected upward. A flap of periosteum is raised from the exposed malleolus, and this subperiosteal dissection is extended distally to free the deltoid ligament down to its fan-shaped insertion on the talus behind, the sustenaculum below, and the navicular in front. It is further freed along its anterior border by incision of the soft parts between the distal anterior angle of the maleolus and the navicular, it being preferable to do this in advance of the deep separation of the deltoid from the medial aspect of the talus. The foot is now brought into a position of forced inversion or varus with special attention to the heel. The periosteal-ligamentous flap is next drawn tautly upward and excised to the extent that it now overlaps the periosteal edge above and this redundant length is removed. The ligament and periosteum are now closed with chromic catgut or kangaroo tendon, and it is well to place two mattress sutures through the deltoid ligament and attach them above as an aid in holding the flap taut during closure. . . . The foot is fixed firmly in marked inversion or varus in plaster of paris, always with emphasis on the heel. . . ."

No weight bearing is allowed for a month. It is imperative that no unsupported walking be done for 6 months. Support during this period is derived from adhesive strapping, shoes with the heels raised on the medial sides or flatfoot braces.

The operation has been employed on patients whose age range is from early childhood to the late thirties with uniformly good results.

R. H. S.

PREVENTION OF POSTOPERATIVE PULMONARY INFECTIONS BY INHALATION OF MICRONIZED PENICILLIN. John A. Dixon and Earle B. Mahoney. *Rocky Mountain Medical Journal 49*:122-125 (Feb.) 1952.

The authors chose 150 cases undergoing operations of the type that are most frequently followed by pulmonary complications for this study. On the day before operation, a nasopharyngeal culture was obtained and plated for penicillin sensitivity and 200,000 units of micronized penicillin was given by inhalation. The day of the operation, 100,000 units was given before operation and another 100,000 as soon postoperatively as the patient was able to cooperate. On each of the first two postoperative days, 100,000 units was given.

Three groups of pulmonary complications were recognized; bronchitis, atelectasis, and bronchopneumonia. In this series, 8 patients had postoperative pulmonary infection (7 had atelectasis and 1 had bronchopneumonia). This constitutes a morbidity of 5.3 per cent compared with a rate of 19 per cent in a series of untreated cases at the same hospital.

The authors conclude that micronized penicillin is an agent of considerable value in the reduction of pulmonary inflammation when used prophylactically in the operative case.

R. H. S.

EXPERIMENTAL STUDIES ON REVERSAL OF THE CIRCULATION IN THE LOWER EXTREMITY. Thomas H. Palmer and C. Stuart Welch. *Surgery, Gynecology and Obstetrics 94:206-214* (Feb.) 1952.

Prompted by the recent revival of interest in using the venous system for revascularization of ischemic organs, the authors have carried out a series of experiments endeavoring to clarify the confusion in regard to the results of attempts at reversal of the circulation in the extremity. In 48 dogs, the femoral vessels were divided and the proximal cut end of the femoral artery was anastomosed to the distal cut end of the femoral vein. In 17 of these animals, the proximal end of the vein was also anastomosed to the distal end of the artery. In the others, these were simply ligated. Anticoagulants were not employed in all cases. The method of Reichert (transection of the soft tissues of the thigh except for the femoral vessels and the nutrient artery of the femur) was employed in the evaluation of the effective circulation after the shunt was performed.

The initial effects were found to be diffuse interstitial hemorrhage, edema, and destruction of tissues distal to the anastomosis. Blood under high pressure flowing centrifugally in the veins ruptures the walls of the capillaries. The collateral venous bed is developed very soon and the blood is quickly returned to the heart through the venous collateral close to the site of the anastomosis. The end effect is closely akin to arteriovenous fistula.

"It appears justified to conclude from these experiments that reversal of the circulation in an extremity is not possible. The procedure of femoral arteriovenous anastomosis is deleterious and should not be applied to eschemic extremities for these already critical limbs will thereby be further deprived of blood supply."

R. H. S.

INTESTINAL OBSTRUCTION. Paul Nemir, Jr. *Annals of Surgery 135:367-375* (March) 1952.

This is the fourth in a series of studies on intestinal obstruction from the Hospital of the University of Pennsylvania. The periods covered in previous reports and the mortality rates reported were: 1905-1922, 30.5 per cent; 1922-1928, 36.3 per cent; and 1934-1943, 11 per cent. The present report covers the period from 1940 to 1950 and includes 430 (all) cases of mechanical intestinal obstruction treated on the surgical service of the University Hospital. Of these, 358 cases underwent operative treatment for the obstruction.

The operative mortality in this group was 10.0 per cent. There was no further decrease in the mortality rate during the part of the period when penicillin and other antibiotics were freely utilized. The mortality rate in

those cases in which gangrenous bowel was found was 31 per cent. Intestinal obstruction due to malignancy accounted for almost one-half of the deaths.

In 8 of the 12 patients who died following operation, dark bloody fluid of the type that has been described in experimental animals by Nemir was found and possibly contributed to the outcome.

R. H. S.

PREOPERATIVE PREPARATION OF THE SKIN WITH A DEPILATORY CREAM AND A DETERGENT. Paul W. Vestal. *American Journal of Surgery 83*:398-402 (March) 1952.

The author has used a depilatory cream of the type currently marketed as a cosmetic preparation for preoperative preparation of the skin in 460 cases. In no instance has there been evidence of irritation of the skin, even in traumatic cases with oozing, abraded skin surfaces. Accidental introduction into traumatic wounds produces no tissue reaction. The cream aids in the removal of detritus and foreign material ground into the skin such as is often found in accident cases. Since the hair is removed in toto and not just above the surface of the skin and since surface detritus is removed, the author proposes that it is probably superior to shaving. In his practice, the use of the depilatory cream was coupled with a pHisohex (Winthrop-Stearns) skin prep.

The cream is without odor. At present, it is subject to tax as a cosmetic. Even so, Vestal does not consider it to be expensive when its merits are taken into account.

R. H. S.